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TUMORS OF THE EXTERNAL AUDITORY CANAL

WITH A REPORT OF ELEVEN CASES

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Tumors of the external auditory canal are so rarely encountered in clinical practice that they are bound to elicit unusual interest. Experience with a number of cases of this type of lesion has prompted me to make a study of the cases which have been observed at City Hospital and at Lakeside Hospital.[1] This has been augmented by a review of most of the recent literature on the subject.

MALIGNANT TUMORS

Squamous carcinoma is the most common malignant tumor of the external auditory canal, and the records reviewed contain 3 cases of this type of lesion, which I am presenting here:

CASE 1.—A white woman, a widow, aged 58 years, presented herself at City Hospital on April 11, 1931 for treatment of a discharging ear. She said that there had been drainage from this ear since she was 4 years old. She had had pain in the ear and the head for about a year. A mass was discovered in the outer canal of the left ear at the first examination in the outpatient department. A biopsy was made, on which the pathologist reported squamous carcinoma, and the patient was advised to enter the hospital for operation.

Physical examination revealed no abnormalities or signs of disease, aside from that in the ear. The right ear showed evidence of a long-standing chronic suppuration which was healed. The left ear was filled with a granulomatous mass which looked like a polyp but was firmer. This mass bled readily and was surrounded by a purulent secretion. The mastoid process on the left side was tender, and there was a questionably enlarged lymph node just beneath the pinna. Two superficial abscesses were evident on the right tonsil, though both tonsils were small. No masses, glands or pulsations were palpable in the neck.

A radical mastoidectomy was performed by Dr. C. E. Engler. Pathologic examination of the material removed from the ear confirmed the diagnosis made at biopsy, squamous cell carcinoma with secondary infection.

It has been impossible to trace the patient since the operation.

1. Dr. Carlos Pitkin gave permission for the use of the cases observed at Lakeside Hospital.

CASE 2.—A Negro, aged 63 years, was admitted to the surgical service of Lakeside Hospital on May 21, 1935. He had been examined in the outpatient department one week earlier for treatment of the right ear. Twenty-five years before, itching had appeared in this ear and had continued for fifteen years. Then the patient had gone to a physician, who had scraped the canal and prescribed some drops for the ears. Soon after that, pus and blood began to drain from the ear, and the discharge had been continuous for the last ten years. Two years before admission, the patient noticed some loss of hearing in that ear. One week before, he had become dizzy.

At the time of the examination, the right ear was discharging a sanguineous, purulent, foul fluid. There was tenderness over the mastoid process. The skin of the right canal was eroded, the entire middle ear cavity was necrotic and the mastoid cells were destroyed. There was absolutely no hearing on the right side. Roentgenographic examination showed extensive destruction of bone throughout the entire right mastoid area, involving the auditory canal and extending into the mastoid antrum. The destruction extended also into the sphenoid bone and involved the lateral portion of the right cranial fossa. Biopsy of the skin of the right auditory canal proved that the growth was a squamous carcinoma.

No operation was performed, but several roentgen irradiations were administered. The patient was acutely ill during most of his stay in the hospital, and at times delirious or unconscious. He died one week after admission.

Necropsy was performed, and the diagnosis of squamous cell carcinoma of the external auditory canal was confirmed. There was also metastatic invasion of the brain.

CASE 3.—A white man, aged 31 years, came into the outpatient department of Lakeside Hospital complaining of a discharge from the left ear. The ear had been draining since he was 2 years old, and hearing was completely absent on the left side. There had been a growth in the left auditory canal for the last five months associated with considerable pain. Four attempts to remove the lesion had been made. On each occasion the pain was relieved but the ear failed to heal properly, and the growth, with its accompanying pain, recurred within a short time. A pathologic report on a biopsy of tissue taken at the last operation had been carcinoma.

The patient had no complaints or symptoms except those relating to the left ear, and physical examination revealed no other abnormalities. A foul purulent discharge was drained from the left ear. A red mass at the junction of the bony and the cartilaginous canal obscured the view of the tympanic membrane. There was no hearing by air conduction, and bone conduction was poor. No lymph nodes were palpable in the region of the left ear. Roentgenograms showed a tumor in the left external auditory canal, with considerable bony destruction around it.

The patient was admitted to Lakeside Hospital and was operated on by Dr. Carlos Pitkin on April 1, 1939. An extensive postauricular incision was made on the left, through the skin and periosteum. The periosteum was elevated, and a self-retaining retractor was inserted. An incision was then made around the entire circumference of the external auditory canal, including a part of the tragus, and just inside the external auditory meatus. This incision was carried through the posterior wall of the canal, and the ear was retracted well forward. The tumor mass in the external auditory canal was removed in one large piece down to the wall. The posterior wall was taken down by means of gouges, and the antrum was entered. This contained granulations but no cholesteatoma. The bridge was removed, and an area suspected of containing tumor tissue was removed from the posterior wall of the canal. There was no evidence of disease in the middle

ear. A portion of the malleus was removed, but the incus was not found. Two specimens of tissue were removed from the remaining portion of the anterior wall in an effort to determine whether all the tumor had been taken. The postauricular wound was left wide open, and this and the external auditory canal were packed with iodoform gauze.

Microscopic examination of the tumor showed irregular foci of compact malignant epithelial cells. The tumor cells, which were of irregular polygonal shape, had large vesicular nuclei and abundant poorly defined cytoplasm. There was moderate pleomorphism, and many of the nuclei were irregular, densely chromatic or slightly elongated. Occasional mitotic figures were observed. No intercellular bridges or keratohyaline pearls were present, but occasional foci of individual cells were suggestive of keratinization. The tumor cells were embedded in irregular and whorling groups of young fibroblasts, with elongated vesicular nuclei. There was an abundance of intercellular collagenous substance along the edges of the tissues. There were also some foci of inflammation with infiltration of the lymphocytes. The pathologist's diagnosis was squamous cell carcinoma.

Biopsy showed no evidence of malignant disease in the specimens of tissue taken from the anterior wall of the canal, after the tumor had been removed.

The patient made an uninterrupted recovery from the operation and left the hospital on the eighth day. He was then referred for radiation therapy.

The radiation was administered by Dr. Eugene Freedman as follows: Chaoul (contact) irradiation and external irradiation were carried out simultaneously. The chaoul contact tube, used at 60 kilovolts and with copper filtration, was applied directly to the central portion of the operative bone defect. Radiation was administered daily, from April 14 to May 10, 1939, in doses of 400 to 600 roentgens. A total of 6,200 roentgens (measured in air) was given. In addition, two external fields, each measuring 7 by 5 cm., anterior auricular and posterior auricular, were irradiated. Each of these areas was treated on alternate days as follows: The voltage was 220 kilovolts, with filtration of 0.5 mm. of copper and 1 mm. of aluminum plus the inherent filter. The individual dose applied over each field was 250 roentgens, measured in air. A total dose of 3,100 roentgens was administered to each of the two external fields. The estimated erythema dose to the tumor area was 12 erythemas.

In April 1940, a year after treatment, the patient had no pain or discomfort and had been working steadily as a machine operator since August 1939. The wound, however, was not completely healed. A small opening, 3 cm. by 4.5 mm., persisted in the postauricular region. There also was evidence of low grade osteomyelitis of the petrous bone, with occasional discharge of small splinter-like sequestrums. There was no sign of recurrence of the tumor.

Incidence.—Although most authors have stressed the rarity of malignant tumors of the external auditory canal, Scott and Colledge [2] made the statement that they believed such lesions are not so rare as usually supposed. They found 70 cases reported in the transactions of the Section of Otology in *Proceedings of the Royal Society of Medicine* during the last thirty-one years. Fraser [3] reported for malignant disease

2. Scott, P., and Colledge, L.: Discussion on Malignant Disease of the Ear (Excluding the Pinna), Proc. Roy. Soc. Med. **32**:1087-1104 (July) 1939.

3. Fraser, J. S.: Malignant Disease of the External Acoustic Meatus and Middle Ear, Proc. Roy. Soc. Med. **23**:71-77 (June) 1930.

of the external auditory canal an incidence of 0.464 per cent observed in private practice and of 0.197 per cent at the Royal Infirmary of Edinburgh. Robinson [4] reviewed 19 cases of malignant tumors of the external ear and auditory canal occurring among 212,000 patients with aural disease seen at the Manhattan Eye, Ear and Throat Hospital. Richter [5] reported only 5 instances of malignant disease of the external auditory canal among 35,000 patients seen at the Erlanger Clinic from 1919 to 1931.

The age of patients with malignant tumors of the external auditory canal corresponds to that of patients with malignant growths elsewhere in the body. Instances have been recorded of occurrence in patients from youth to extreme old age, but the majority of patients are in the fifth or the sixth decade of life. The cases of malignant disease of the external auditory canal recorded in the literature seem to be evenly divided between men and women.

Pathology.—The most important types of malignant disease of the external auditory canal are: (1) carcinoma, (2) sarcoma, (3) rodent ulcer and (4) endothelioma.[2] As already mentioned, the most common malignant growth is squamous carcinoma,[6] although a number of cases of adenocarcinoma [7] have been observed. Several cases of prickle cell carcinoma [8] have also been reported. Jeschek [9] reported a case of

4. Robinson, G. A.: Malignant Tumors of the Ear, Laryngoscope **41**:467-473 (July) 1931.

5. Richter, H.: Zur Klinik, pathologischen Anatomie und Therapie der bösartigen Geschwülste des Ohres, der oberen Luft- und Speisewege (nach den Erfahrungen der Erlanger Klinik aus den Jahren 1919 bis 1931), Ztschr. f. Hals-, Nasen- u. Ohrenh. **31**:169-192, 1932.

6. (*a*) Barnes, E. B.: Carcinoma of the Ear, Proc. Roy. Soc. Med. **23**:67-70 (June) 1930. (*b*) Risch, O. C., and Lisa, J. R.: Primary Carcinoma of the External Auditory Canal and Meatus: Review of Literature and Report of Case, Laryngoscope **48**:668-681 (Sept.) 1938. (*c*) Land, F. T.: Epithelioma of the External Meatus: Case, J. Laryng. & Otol. **50**:848-851 (Nov.) 1935. (*d*) Fineberg, M., and Jorstad, L. H.: Primary Carcinoma of the External Auditory Canal, Ann. Otol., Rhin. & Laryng. **43**:464-471 (June) 1934.

7. (*a*) Lukens, R. M.: Adenocarcinoma of the External Auditory Canal, Ann. Otol., Rhin. & Laryng. **45**:567-573 (June) 1936. (*b*) Nichinsson, A. G.: Ein Fall einer Epithelgeschwulst des äusseren Gehörganges (Zur Frage über die Pathologie der apokrinen Drüsen), Monatschr. f. Ohrenh. **67**:210-221 (Feb.) 1933.

8. (*a*) Denison, W. C.: Prickle Cell Epitheliomata with Involvement of Facial Nerve in Tympanum, Laryngoscope **44**:927-930 (Dec.) 1934. (*b*) Blackwell, H. B.: Report of Two Cases of Neoplasm in the External Auditory Canal: Operation; Recovery, ibid. **44**:105-108 (Feb.) 1934.

9. Jeschek, J.: Ueber Blutgefässgeschwülste des Gehörganges und des Mittelohres, Monatschr. f. Ohrenh. **70**:1297-1302 (Nov.) 1936.

hemangioendothelioma and Jones [10] a similar case in which the diagnosis was hemangioma with malignant change. A case of myxofibrosarcoma in an 8 year old girl was reported by Hand and O'Connor,[11] who commented that they had found only 2 similar cases in the literature. Adam and Gilmour [12] reported a case of teratoma in the external auditory canal, which they thought had arisen from a squamous epithelioma.

Scott and Colledge [2] suggested that primary carcinoma should be divided into two main types, identified by early or late involvement of the tympanum. Some growths are recorded as arising in the middle ear, but Scott and Colledge cited Ernest West's statement that all squamous carcinomas arise in the fundus of the external meatus and spread inward to involve the tympanum. The meatus appears to be the starting point in the majority of cases. In the second type, that in which the tympanum is not involved until late in the course of the disease, primary ulceration in the meatus usually attracts the patient's attention by causing pain or irritation before the growth has spread widely.

Sarcoma and endothelioma present few clinical features to distinguish them from carcinoma, and the differentiation can be made only by histologic examination.

Symptoms and Diagnosis.—The outstanding symptom of malignant disease of the external auditory canal is pain, which, as Fraser [3] pointed out, is usually worse at night. I am inclined to agree with the opinion stated by Bowman,[13] that pain is not an early symptom of carcinoma of the external auditory canal, or the region of the middle ear or the mastoid, for the size of the tumor mass when pain appears shows that the growth has been present for some time. Nevertheless, pain is the symptom which is most likely to bring the patient to the otologist. Other symptoms are ulceration, aural discharge, deafness, giddiness and in late stages facial paralysis.

Of the 70 patients whose cases were reviewed by Scott and Colledge,[2] 19 had symptoms which immediately aroused suspicion of malignant disease. Of the remaining 51, the majority had otorrhea, with blood-stained discharge in a few cases; 19 had chronic suppurative otitis media with or without vertigo and pain, 8 had furunculosis or meatal eczema, and 15 had facial palsy when first examined by the otologist.

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10. Jones, J. A.: Hemangioma of the Tympanic Cavity, J. Laryng. & Otol. **45**:265-266 (April) 1930.

11. Hand, F., and O'Connor, G. B.: Myxofibrosarcoma of the External Ear, Ann. Otol., Rhin. & Laryng. **47**:1096-1100 (Dec.) 1938.

12. Adam, J., and Gilmour, M. D.: Teratoid Tumour of the External Auditory Meatus, J. Laryng. & Otol. **45**:550-552 (Aug.) 1930.

13. Bowman, R. J.: Carcinoma of the External Auditory Canal, Middle Ear, and Mastoid, Ann. Otol., Rhin. & Laryng. **49**:225-231 (March) 1940.

Many had some degree of deafness. One patient, complaining of pain, was treated for one year for wax before seeing an otologist, who removed the wax scab and revealed an ulcer in the external meatus. Few patients complain of the discharge, because they have become accustomed to it. The onset of pain with irritation and with or without vertigo or deafness brings them to the otologist. Fineberg and Jorstad [6d] stressed the fact that persistent pain out of keeping with the objective findings in the external auditory canal should always be suggestive of a malignant growth.

Other factors which should increase the suspicion of malignant growth are: (1) toughness of the granulations when touched with a probe, (2) recurrence after removal and tendency to bleed easily, (3) necessity of persistent treatment, (4) pain on chewing and (5) granulations or papillomas attached to the deep meatal wall.

The diagnosis of malignant disease of the external auditory canal can be made only by histologic examination. All cases in which granulations or a persistent ulcer are present, especially in patients more than 40 years of age, require consideration of malignant disease. If early diagnoses are to be made, the possibility of malignant growth should be considered also in all cases of long-standing, chronic suppuration of the ears, because the records show that a great proportion of patients have had aural discharge for years.

In the differential diagnosis of malignant disease, one must consider a type of nonmalignant ulcer the clinical appearance of which closely resembles that of a malignant ulcer; the differentiation can be made only by microscopic examination of removed tissue. Scott and Mollison [14] discussed in detail 6 cases of this type of growth in which the ulcer healed slowly under local treatment. They pointed out that nonmalignant ulceration in the external auditory meatus, apart from that produced by closely packed cholesteatomatous masses in the meatus, is not a common condition. It assumes importance because it must be differentiated from early epithelioma. This type of ulcer is generally found on the floor, but sometimes also on the anterior wall or on the posterior inferior wall, of the canal. It shows small granulations and an area of bare bone. The symptoms are foul-smelling discharge and discomfort or pain, usually with deafness, probably due to debris.

I have observed 1 case of this type of disease, in which the ulcer healed completely under local treatment. The report of the case follows:

CASE 4.—A woman aged 43 years had an ulceration of the anterior portion of the external auditory canal. The appearance was that of a furuncle, which

14. Scott, S., and Mollison, W. M.: Cases of Non-Malignant Ulceration in the External Auditory Meatus Resembling Appearance of Early Malignant Disease, J. Laryng. & Otol. **54**:350-353 (June) 1939.

ulcerated but did not discharge pus. Biopsy showed no evidence of malignant disease. The patient was treated for about six weeks. The lesion healed completely and has shown no tendency to recurrence within a year.

Treatment.—The treatment of malignant tumor of the external auditory canal is surgical removal, usually accompanied by radical mastoidectomy. Some authors advocate the radical operation in all cases, and others believe that when there is no involvement of the tympanum a radical procedure is not necessary and that the growth can be removed through the meatus. Some form of irradiation, either with radium or with roentgen rays, is usually employed after surgical intervention.

Pfahler and Vastine [15] pointed out the difficulties of irradiation therapy in this region. They said that a greater proportion of failures occur in the treatment of cancer of the ear than in that of the disease in any other relatively superficial part of the body. They expressed the belief that most of the failures are due to faulty or insufficient early treatment. They recommended that in the treatment of malignant disease of the external auditory meatus, the growth be completely removed by dissection or curettage and that then a capsule of radium sufficient to destroy the surface disease be applied. This usually involves a 50 mg. capsule in 1 mm. of platinum, which should be left in place for three hours. When it is impracticable to introduce the radium capsule, high voltage roentgen rays, with filtration of not less than 2 mm. of copper, are used. Even when radium is used in the auditory canal, sufficient external irradiation, either with radium or with high voltage roentgen rays, should be added.

LOCALLY RECURRENT GROWTHS

In addition to the cases of true malignant disease, there is a group of cases in which the growth does not show a tendency to mitosis or metastasis and hence from the pathologist's standpoint is not malignant, yet it continually recurs and from the otologist's point of view is as troublesome to deal with as a malignant growth.

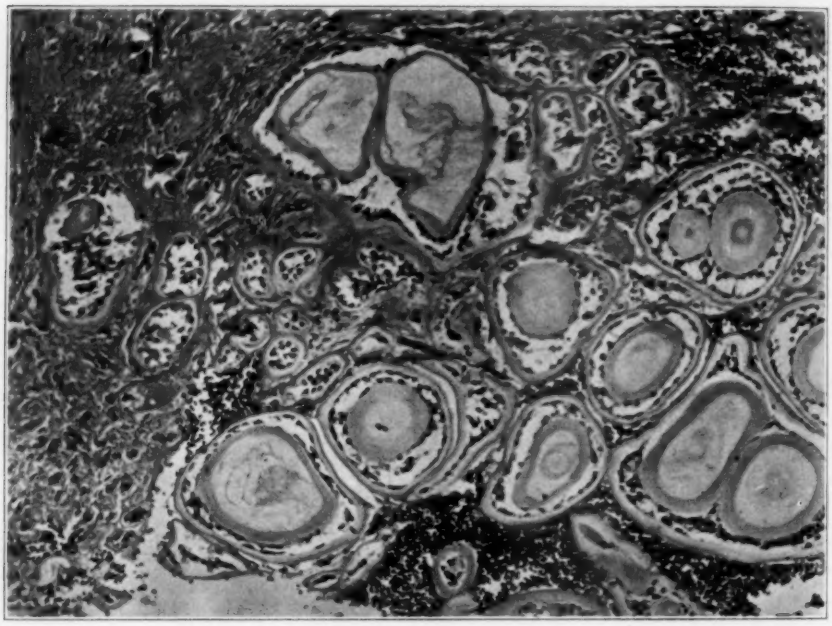
In our experience at City Hospital, my associates and I have encountered 2 cases of this type. In the first, a diagnosis of cylindromatous lymphangioma was made by Dr. Howard T. Karsner. So far as I have been able to determine, no other case of this type has been recorded in the literature. The report of this case is as follows:

Case 5.—A Negress aged 45 years was admitted to City Hospital in October 1939 for treatment of pain in the left ear. Ten years previously she had received

15. Pfahler, G. E., and Vastine, J. H.: Treatment of Cancer in the Region of the Ear, Am. J. Roentgenol. **37**:350-357 (March) 1937.

a blow over the left ear, and a little later she had noticed loss of hearing on that side. There was also a small growth on the floor of the auditory canal, which caused pain at first but ceased to bother her after a short while, and she did not seek medical advice regarding it until several years later. In 1936 the growth was removed by cautery, and the histologic diagnosis was cylindroma.

Three years later a similar growth appeared in the roof of the canal, and the patient returned to the hospital for treatment. The lesion, which was soft and movable, was attached to the roof of the canal just inside the orifice and almost filled it, obstructing the view of the tympanum. Bone conduction was good, but air conduction impaired. The tonsils were enlarged, as were the cervical glands below both angles of the mandible.



Photomicrograph of cylindromatous lymphangioma (case 5), to show lymphangiomatous arrangement of cells of endothelial type and cross sections of hyaline cylinders. (Hematoxylin and eosin; × 117.)

At operation the large mass of tumor, which was not encapsulated, was encountered in the external auditory canal. It invaded the skin of the canal but did not involve the bone. The tumor, together with the membranous portion of the external auditory canal, was removed. A plastic procedure was carried out on the stump of the membranous canal, similar to that employed in a radical mastoid operation. Iodoform gauze packing was inserted into the external auditory meatus, and the postauricular incision was closed with clips.

The pathologic diagnosis, made by Dr. Karsner, was cylindromatous lymphangioma. His report of the microscopic examination of the tissue was as follows:

"There is a partially covering layer of stratified squamous epithelium with a slight excess of superficial keratinized cells. Beneath this is a mass of densely collagenous connective tissue which contains several nodules 1 to 3 mm. in diameter.

Some of these are sharply defined, and others fuse with the surrounding tissues. Two different types of growth are present. In some of the nodules there is a hyalinized connective tissue matrix in which are nests of cells, generally rounded but occasionally stellate with short cytoplasmic processes. The nuclei are large and vesicular, but there are no mitotic figures. In some there is a central lumen, either empty or containing acidophilic granular precipitate; there is no blood. In numerous places the cells are arranged in sleevelike fashion about acidophilic hyaline masses, evidently cylindric and irregularly or concentrically laminated. The cells line the surrounding space so that they are applied to the peripheral wall and to the cylinders with an empty space between the two layers. There are small hemorrhages in the tissue, evidently traumatic.

"The essential cells have the general features of endothelium, and the presence of spaces, devoid of blood, indicates that the basic lesion is lymphangioma. The production of hyaline cylinders justifies the term cylindromatous lymphangioma. There are a few simple epithelial glands, in no way connected with the cells of the tumor. There is no squamous epithelium, other than that on the surface, and this fact, together with the other features, excludes cholesteatoma. The general features are not like those of salivary gland mixed tumor." The accompanying figure illustrates the structure of the lesion.

After the operation there was considerable drainage from the ear, and the patient complained for a time of pain and giddiness. Three months later the ear was completely healed, and there was no evidence of recurrence of the tumor. However, just three weeks after this examination the patient returned, complaining of some discomfort in the ear, and there appeared to be renewed activity of the growth. She received irradiation treatments, and one year after operation she apparently was entirely well.

The condition in the following case also proved extremely troublesome to treat and is unlike any recorded in the literature.

Case 6.—A married Negress, aged 39 years, was first seen in the otolaryngologic department at City Hospital in February 1937. She had been treated in the hospital a few months previously for burns caused by lye and returned at this time because there was a discharge from the right ear. Examination revealed atresia of the right auditory canal with granulations and discharge. The tympanic membrane appeared dull and thickened. A roentgenogram showed haziness throughout the right mastoid area.

The patient was seen again in January 1938, at which time the right external ear was completely occluded by a keloid growth. She was advised then to enter the hospital for a radical mastoidectomy but did not do so until eight months later (August 1938). At that time the patient said that she had suffered excruciating pain in the ear and the head for the last six months. The few days before she entered the hospital the pain had been even more severe, and there was tenderness over the mastoid process, headache and discharge from the ear. The patient also complained that she had had difficulty with her eyes since the onset of the trouble in the ear.

A scar and a keloid were present, beginning in the midline on the neck beneath the chin and running up to the right ear and the head. The auditory canal of the

right ear was completely closed with scar tissue. The mastoid area was tender to pressure. There was some discharge from paracentesis which had been performed previously for the drainage of the canal.

A radical mastoidectomy was performed on Aug. 26, 1938. Because of the contracture of the external auditory canal by scar tissue, it was difficult to make a proper flap for the new canal, but a T-shaped opening was made and fragments of skin were sutured to the periosteum. The posterior incision was closed with clips. A gauze pack treated with petrolatum was placed in the new canal to keep it patent.

After the operation the patient returned for treatment again and again, because of occlusion of the auditory canal by keloid. Despite repeated packing of the canal to keep it patent, scar tissue continued to form, and finally the canal was again completely occluded. Numerous consultations were held as to the advisability of additional surgical treatment, and the patient was also referred to the dermatologists for suggestions. An attempt at radiation therapy was made, but without beneficial effect.

Finally, about a year after the mastoidectomy, the patient was advised to return to the hospital for a plastic operation but failed to do so and has not been seen at this hospital since September 1939.

Löwy [16] discussed 2 cases in which, as in those just presented, the histologic examination showed no sign of malignant disease and yet the growths were locally recurrent. His first case was of fibroma in a man aged 63 years. The patient complained of no pain, and there was no secretion from the ear. The growth had been removed four years earlier but had recurred, and so irradiation therapy was administered. In the other case the patient was a woman, aged 28 years, who complained of a feeling of fulness in the right ear and had had some headache. A polyp-like tumor was removed, which showed a tendency to recur immediately. The lesion had to be removed several times. The pathologist reported that it was a benign growth (pseudomyxomatous fibroma), but in view of the striking tendency to recurrence irradiation therapy finally was advised. After the administration of 555 milligram hours of radium, the patient had no recurrence for two years.

BENIGN TUMORS

The most common benign tumor of the external auditory canal is osteoma, and the records reviewed contain 5 cases of this type of lesion. In 2 of these no operation has been performed, but 1 is of special interest, in that there are osteomatous growths in both ears. A summary of the 5 cases follows:

Case 7.—A white man aged 28 years, an employee of City Hospital, came to the otolaryngologic clinic on Oct. 20, 1939, complaining of a feeling of stiffness in the left ear, which he said had been present for about twenty-four hours.

16. Löwy, K.: Zur Pathologie der Tumoren des äusseren Gehörganges, Monatschr. f. Ohrenh. **70**:1319-1324 (Nov.) 1936.

In the right ear there was a small, bony, hard mass, about 2 to 3 mm. in diameter, on the tympanic ring just anterior to the short process. In the left ear there was a similar but slightly larger growth. A diagnosis of bilateral osteoma of the external canal on the tympanic ring was made.

The patient was examined again three months later, at which time his only complaint was bilateral tinnitus. He is being kept under observation.

CASE 8.—An Italian laborer aged 47, married, was admitted to Lakeside Hospital on Nov. 9, 1939, for surgical treatment of bilateral inguinal hernia and external and internal hemorrhoids.

Examination disclosed a tumor of the right ear, which obstructed the right external auditory canal and was diagnosed clinically as an osteoma. The growth appeared as a pale, meaty-looking, rounded mass in the middle portion of the right external canal, and it obscured the drum. There was no reduction of hearing on this side, and the patient said he had not had otorrhea, otalgia or tinnitus.

The tumor was not removed, but the patient was advised to return to the otolaryngologic clinic for additional observation and treatment.

CASE 9.—A married woman aged 20 years was admitted to Lakeside Hospital on July 9, 1938 for treatment of exostosis of the osseous meatus of the left ear. The obstruction of the canal of the left ear had been gradually developing since birth. The growth appeared to be a small rounded bony mass, attached to the anterior wall. This was removed by excision, and the patient was discharged the same day.

The specimen removed consisted of a small gray calcareous nodule of tissue, 5 mm. in its greatest diameter. Histologic examination showed that the nodule was covered by a thin layer of stratified squamous epithelium. The epidermis showed no abnormality, and beneath it the corium was fibrous. The bulk of the nodule was comprised of well differentiated bone, represented by fairly plump trabeculae separated by a scant marrow. There was no evidence of aplastic hyperplasia. The pathologist reported that the histologic observations were consistent with the clinical diagnosis of exostosis.

CASE 10.—A laborer aged 20 years was seen in the outpatient department of Lakeside Hospital in December 1932, complaining of frequent sore throats. An osteoma blocking the right external canal was discovered, and removal was attempted on Jan. 3, 1933. At that time the growth was only partially removed, because of its broad base. The patient said that there had been loss of hearing in the right ear during the last six months but that he had had no pain or discharge from this ear and no tinnitus. After the first attempt at removal, the right external auditory canal was still partially blocked by a bony projection from the posterior wall of the canal. The tympanic membrane could be seen.

At operation, performed by Dr. Chamberlain on Jan. 20, 1933, a postauricular incision was made and the skin and periosteum were elevated. The mastoid cavity was entered with gouges. A large osteoma was found, almost completely filling the external auditory canal and extending inward to the middle ear cavity. As the posterior wall of the canal was removed, the middle ear was entered from behind and was seen to be filled with cholesteatoma. No trace of membrane or ossicles was found and no evidence of mastoid antrum or aditus. The mastoid cells were large and apparently normal, and no attempt was made to exenterate them. The facial nerve was uncovered and recognized but was not cut during the removal of the posterior wall. Nevertheless, a paralysis of the right facial

nerve, with some ptosis of the upper lid and definite nystagmus, developed after the operation. The patient also displayed symptoms of labyrinthitis. The latter subsided, although the paralysis of the facial nerve persisted.

Histologic examination of the tissue removed at operation showed that it was comprised of a mass of bone, the overlying periosteum, subcutaneous tissue and epithelial covering. There was a small amount of hemorrhage and mononuclear infiltration in the subcutaneous tissue as well as some vascularization of bone. No definite pathologic diagnosis was made.

CASE 11.—A married woman aged 48 years complained of loss of hearing. Examination at Lakeside Hospital revealed a tumor in the left auditory canal. The deafness had begun five years earlier and had steadily grown worse. The patient had never had pain or aural discharge until a physician elsewhere treated her with radium several months before.

The growth was removed with the patient under nitrogen monoxide anesthesia, and the tympanic membrane was visible after removal of the mass. Even at operation, however, the exact situation of the tumor was doubtful, and no definite clinical diagnosis was made.

The specimen consisted of several small pieces of tissue, the largest about 0.7 cm. in diameter. The report of the microscopic examination was as follows: "The nodular mass is covered by stratified squamous epithelium, which in places is attenuated to a thickness of only a few layers. Ulceration is present, beneath which there is infiltration of polymorphonuclear leukocytes and lymphocytes, and a somewhat deep necrosis of the underlying tissue. The main mass of the lesion is made up of irregularly whorled connective tissue, in places almost wholly fibrillar and in other places rich in nuclei of large spindle form of the young type of cell. There are no mitotic figures. Thin-walled blood vessels are scanty and have their own endothelial lining. There are several small masses of calcified material with poorly defined lamellas, canals and bone cells. These masses are irregularly outlined and in intimate continuity with the surrounding tissue. Cells of the type of osteoblasts are found in small numbers, but there are no osteoclasts. Thus, this represents not destruction but rather production of bone." The pathologic diagnosis was osteofibroma.

According to Banyai and Janota,[17] osteoma of the auditory canal appears usually after puberty and is almost always unilateral. The incidence is much greater in males than in females. According to Körner it occurs three to five times as often in men as in women, and according to Bezold, eleven times as often. The reason for this predominance is unknown. The incidence also is greater in northern lands, which suggests that there may be a metabolic factor in the causation.

Lillie and Williams[18] pointed out that osteoma shows no tendency to recur and that whenever possible the growth should be removed through the auditory meatus. In some cases, however, the size and position of the tumor demand a more radical surgical procedure.

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17. Banyai, J., and Janota, F.: Ein mandelförmiges obturierendes Osteom des äusseren Gehörganges, Monatschr. f. Ohrenh. **67**:983-984 (Aug.) 1933.

18. Lillie, H. I., and Williams, H. L., Jr.: Osteoma of the External Auditory Canal, S. Clin. North America **11**:801-808 (Aug.) 1931.

Other benign growths of the external auditory canal which have been reported in the literature include xanthoma,[19] dermoid teeth [20] Recklinghausen's disease,[21] pigmented and nonpigmented nevi [16] and organized blood clot.[8b]

INFLAMMATORY LESIONS

Polyps in the external auditory canal are not uncommon in cases of chronic infection. The records of Lakeside Hospital include 4 cases of this condition in children. In 1 of these instances the middle ear was filled with a large cholesteatoma. These cases will not be reported in detail. Attention is called to them, however, because of the possible significance in relation to neoplastic disease. Certainly the case reported by Pattee [22] suggests that this possibility cannot be completely ignored. His patient was a man aged 60 who had a growth in the right ear which appeared to be a large polyp, 2.5 cm. in diameter, protruding from the right external auditory canal. A foul purulent discharge filled the canal, and a second polyp was seen on the floor of the canal in the region of the isthmus. The growth had first appeared in the patient's infancy but had become black and dropped out when he was 18 years old. It soon reappeared and had been present since. The growth was removed through the external auditory canal, and pathologic examination showed that the outer third and the pedicle consisted of myxedematous fibrous tissue. The middle third was of a different color and appearance and was found to be a cystadenoma which showed no evidence of malignant change.

That chronic infection has some relation to the development of neoplastic tumors is suggested also by the fact that a preponderant number of patients with malignant disease of the external auditory canal have had aural discharge for years. This fact may have considerable clinical importance from the standpoint of diagnosis and suggests that the possibility of malignant disease should be kept in mind in the consideration of all cases of chronic suppuration of the ear and of all cases of polyposis. Many malignant lesions of the auditory canal have appeared clinically as simple polyps, and only histologic examination has revealed their true nature.

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19. Rosenberger, H. C.: Solitary Xanthoma of the External Auditory Canal, Arch. Otolaryng. **26**:395-399 (Oct.) 1937.

20. Marshall, G. G.: Dermoid Teeth in the External Auditory Canal, with Comments on Teratomas and Dermoids in General, New England J. Med. **214**: 202-204 (Jan. 30) 1936.

21. Wieder, H. S.: Von Recklinghausen's Disease, with Report of Case Involving the External Auditory Canal, Laryngoscope **42**:364-370 (May) 1932.

22. Pattee, G. L.: Unusual Tumors Seen in Otolaryngological Practice: Aural Polypus with Cystadenoma, Colorado Med. **32**:719-720 (Sept.) 1935.

If patients themselves were educated to the potential seriousness of continuous discharge from the ear and if otologists were more alert to suspect malignant disease, it might be that in a greater proportion of cases malignant tumors of the external auditory canal would be discovered and treated early. This would, no doubt, improve the prognosis in these cases considerably, for, as Scott and Colledge,[2] emphasized, the rate of growth of auditory tumors is slow and the lymphatic spread often late.

SUMMARY

Eleven cases of tumor of the external auditory canal from Cleveland City Hospital and Lakeside Hospital are reviewed. In this series, there were only 3 cases of malignant tumor. In all of these the lesion was squamous carcinoma; 1 patient was a white woman, 1 a Negro and the other a white man.

Two cases of pathologically benign but locally recurrent tumors are perhaps the most interesting from the clinical point of view. One of these was of a cylindromatous lymphangioma, in a Negro woman. No other case of this type has been reported in the literature. The other case was of persistent keloid formation which blocked the external auditory canal. The patient in this case also was a Negro woman.

One case of a benign ulcer resembling a malignant lesion is described.

Five cases of osteoma also are included.

The recent literature on tumors of the external auditory canal is reviewed.

SUPPURATION OF THE PETROUS PYRAMID

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The subject of infection of the petrous pyramid has been discussed among otologists for a sufficient time so that the typical, clearcut condition is now rather easily recognized. Otologists are now what might be called "pyramid minded" and are on the look-out for this condition, rather than being surprised when it is encountered.

The symptoms in the typical case have been repeatedly stressed: pain in the region of the eye, discharge from the ear or, its equivalent, from the mastoid wound, and some degree of sepsis. These are regarded as the cardinal signs; and yet how often is the typical case encountered? A review of the records of the last ten years shows that in approximately 50 per cent of the cases these signs are present and that in the remainder there is deviation from this pattern, often to a considerable degree. One should not expect all the signs to be present in any single case. In a large percentage of cases there are signs of pyramid involvement subsequent to mastoidectomy, the interval varying from a few days to several months. However, it should not be forgotten that the infection in the pyramid occurs at the time of the invasion of the middle ear. In some cases the signs of pyramid involvement are present at this early stage, but in by far the great majority the signs either are mild or are overshadowed by the middle ear and mastoid symptoms. Infection of the pyramid should be regarded as a complication of the middle ear process, rather than one of the mastoid.

Regarding the pain, it may be stated that there is no doubt that in many cases congestion or infection is present in the pyramid at the time of the involvement of the middle ear, yet gives no symptoms during the disease and resolves when the infection of the middle ear or mastoid resolves under appropriate treatment.

Infection in the bone itself does not imply pain. If the infection is purely within the bone, it is not until the anomaly causes pressure within the bone itself that pain is felt.

The typical pain is in or behind the eye. It is generally regarded as due to irritation of the first branch of the trifacial nerve, although there are some who regard it as due to irritation of the greater superficial

Read before the Eastern Section of the American Laryngological, Rhinological and Otological Society, Pittsburgh, Jan. 5, 1940.

petrosal nerve, which is in close relation to the pyramid. In either case, these are structures outside the pyramid, and they are not affected by the infection until the inflammation extends beyond the bone itself. A considerable part of the clinical picture is produced by the involvement of structures outside the bone.

By far the most common complaint is generalized frontal pain, usually unilateral and confined to the affected side; it is dull and disturbing, rather than sharp, and is apt to be intermittent and worse at night. This pain, as is known, occurs in association with many aural conditions, so that its presence is not diagnostic except in conjunction with other signs. Many patients, because of either age or mentality, are unable to localize the pain definitely. Pain in the side of the face and in the teeth ascribed to involvement of the other branches of the fifth nerve is occasionally complained of and should be accorded the same importance as the pain located higher. Pain in some part of the head or face is present in almost every case. It is the most constant of all the symptoms of infection of the pyramid.

Of the other cranial nerves, the sixth is most frequently involved, and the external paralysis of a rectus muscle, associated with other signs, is a valuable indication of the process. The paralysis often occurs late and may be partial rather than complete, amounting simply to a weakness of the muscle. The time of the onset of this paralysis is of the greatest importance, for if it occurs a considerable time after the onset of the acute infection it is almost diagnostic of involvement of the pyramid. Paralysis of the seventh nerve occurs with relative frequency. Involvement of this nerve indicates a process occurring within the bone itself, usually in the perilabyrinthine region; although the involvement may be complete, it is more often only partial and in many instances only temporary, the paralysis resolving in spite of the progressing infection. Thus, a history of a facial weakness, even if the condition is not present at the time of the examination, should be regarded as significant.

Many patients present symptoms of infection of the pyramid at the time of the involvement of the mastoid, which completely clear up after a simple mastoidectomy. In others the symptoms apparently clear up only to reappear. Still others present no advance warning until signs of meningeal irritation appear. A case illustrating the last of these circumstances is reported as follows:

REPORT OF CASE

CASE 1.—E. L., a boy aged 8 years, was admitted to the hospital on June 9, 1939 with a history of a mastoidectomy on the right side on May 4. He had made an apparently uneventful recovery and was discharged from the hospital on May 11.

Because of a slight increase in temperature and continued discharge from the ear, he received roentgen therapy, but at no time did he complain of pain or headache.

At the time of his admission the patient complained of persistent headache and pain behind the right eye, which had existed for twenty-four hours. There was slight weakness of the right external rectus muscle. On admission the temperature was 100.2 F., but it quickly rose to 104 F., and during the night the patient vomited

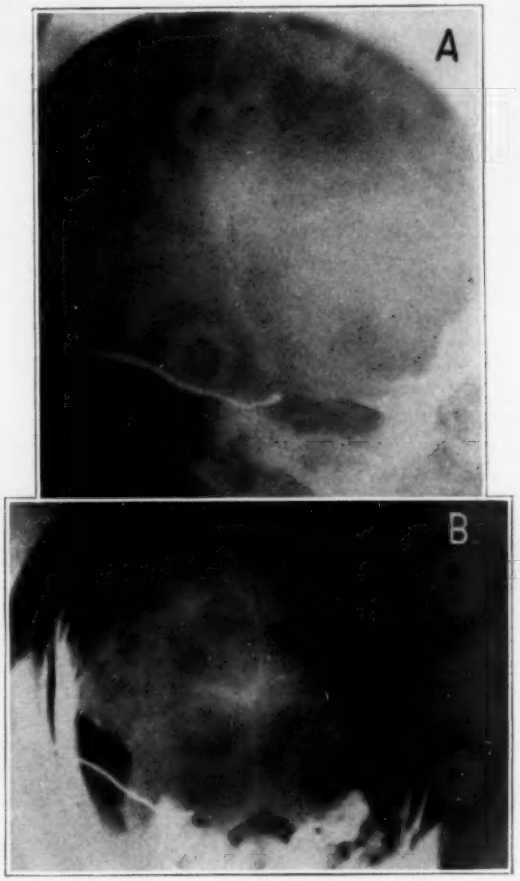

Fig. 1.—Roentgenograms of the patient in case 1: *A*, with the patient in the Stenver position and a probe in the petrous pyramid, and, *B*, with the patient in the modified base position and a probe in the petrous pyramid.

for the first time. In the morning he had a stiff neck; there was a positive Kernig sign, and a spinal tap showed the fluid to be under pressure and the cell count to be 6,600. The roentgenogram revealed definite infiltration, and there was apparently some decalcification.

An operation on the pyramid was performed on June 10. The solid angle was removed down to the labyrinth. No fistula was found. The pontile cistern was drained. On elevation of the dura of the middle fossa, pus was found internal to

the labyrinth, and an opening into the bone could be demonstrated. Culture of the pus showed hemolytic streptococcus. The boy made an uneventful recovery and was discharged on August 19.

Comment on Case 1.—Most clinicians have recognized both the acute and the chronic type of petrositis, the chronic type, as a rule, being one which has become quiescent except for a persistent discharge but in which the acute signs may reappear at any time. However, Lillie and Williams [1] reported 6 cases of chronic suppurative lesions of the pyramid, in 4 of which there was no discharge from the ear and in 1 no pain whatever. That an infectious process may be dormant in the pyramid for months or even years must be recognized.

While discharge from the ear is one of the usual signs, many patients are seen who show no discharge. There has been an attempt by some writers to draw conclusions from the amount and duration of the discharge and from the rate of decrease. I feel that this sign is not of great importance except in relation to the pain, a decreasing discharge associated with a subsidence of pain being the natural condition in favorable progress, and that only in the presence of increasing pain is a lessening of the discharge of great significance.

The history of an aural infection or mastoidectomy occurring many months before with an apparently good recovery should not be lightly passed over. In a paper read before the American Laryngological, Rhinological and Otological Society two years ago, I reported [2] a case in which the infection of the middle ear occurred in March and the infection of the pyramid remained quiescent, with a dry ear, until the following November.[2] The report of another case in this category follows:

CASE 2.—B. W., a girl aged 11 years, was first seen Dec. 24, 1938, with the following history:

After scarlet fever she had been admitted in July to a hospital for patients with contagious disease, having earache on the left side, fever, headache and vomiting of two days' duration. The temperature at that time was 103 F. and the pulse rate 110. She presented a stiff neck; there was a positive Kernig sign, and the left ear drum was bulging. The spinal fluid was cloudy and contained about 750 cells. The patient was given large doses of sulfanilamide. On the ninth day of hospitalization paralysis of the left external rectus muscle developed. Three days later culture of the spinal fluid showed a nonhemolytic streptococcus. On the fifteenth hospital day paralysis of the left facial nerve appeared and a simple mastoidectomy was performed. There was little destruction of the mastoid cells, and they contained a thin

1. Lillie, H. I., and Williams, H. L.: Chronic Suppurative Lesions of Petrous Pyramid: Report of Six Cases with Different Pathologic Characteristics, Arch. Otolaryng. **29**:345 (Feb.) 1939.

2. Moorhead, R. L., and Baker, J. P.: Suppuration of Petrous Pyramid: When and How to Operate; Report of Thirty Cases, Arch. Otolaryng. **28**:497 (Oct.) 1938.

mucoid discharge. Both the dura and the lateral sinus were exposed and appeared normal. Seventeen days after the appearance of the paralysis of the left external rectus muscle and eleven days after the mastoidectomy, the paralysis disappeared; the temperature gradually declined and the patient, thought to have recovered, was discharged from the hospital forty-two days after admission. The paralysis of the facial nerve slowly cleared up, and the mastoid wound healed, the middle ear becoming dry. On December 9 the child was again admitted to the hospital, complaining of headache, earache on the left side and vomiting. The left ear drum was dull, but there were no bulging and no swelling of the mastoid scar. The tem-

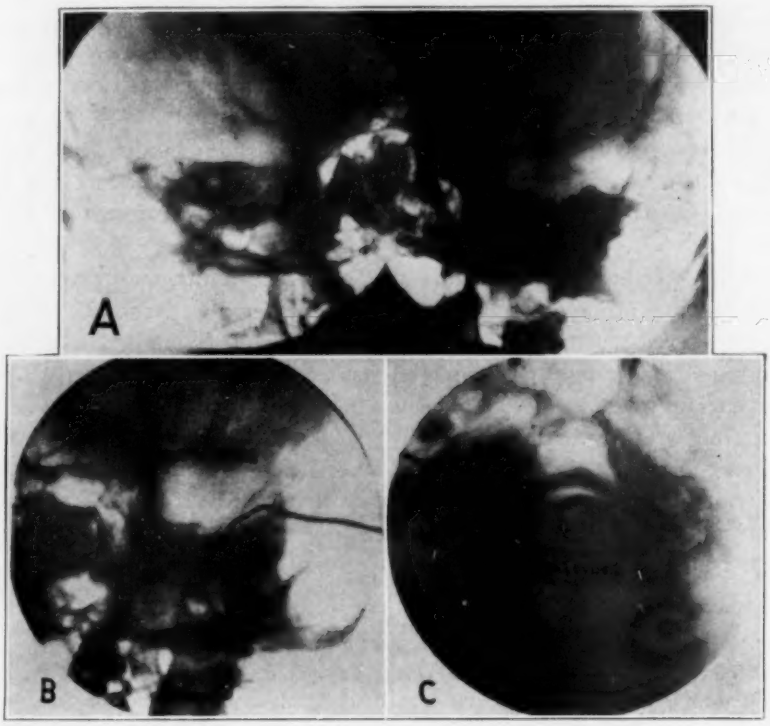

Fig. 2.—Roentgenograms of the patient in case 2: *A*, with the patient in the Stenver position, showing erosion of the cortex; *B*, with the patient in the Stenver position and a probe in the petrous pyramid, and, *C*, with the patient in the Taylor position and a probe in the petrous pyramid.

perature was 102 F. and the pulse rate 120. A lumbar puncture revealed a cloudy spinal fluid under increased pressure, with 1,800 cells. A direct smear showed one chain of gram-positive cocci. The white blood cell count was 13,600. Roentgen examination showed the petrous ridge on the left to be irregular, and apparently there was an erosion on its upper margin. Massive doses of sulfanilamide were again administered. On December 24 I saw the patient, and an operation on the pyramid was performed. The solid angle was removed down to the labyrinth, but no fistula was found. The pontile cistern was drained, about 50 cc. of spinal fluid

being removed. On elevation of the dura, free pus was encountered internal to the labyrinth, and a considerable area of bony necrosis was found on the anterior surface of the pyramid. Rubber drains were placed in the cavity of the bone, and the wound was left open. The child made an uneventful recovery.

Comment on Case 2.—The infection of the petrous pyramid probably was present during the patient's first hospitalization, inasmuch as she had headache and paralysis of both the sixth and the seventh nerve together with meningeal irritation. Thus, the infection remained dormant from August to December without manifesting itself, and causing no discharge from either the canal or the mastoid wound. A simple mastoid operation plus the administration of sulfanilamide temporarily halted the process; the paralysis of the facial and the sixth nerve disappeared, and the patient was thought to have recovered.

In these days of almost universal use of sulfanilamide, may it not be well to remember that perhaps the underlying factor is being controlled, rather than cured, and that the disease may reappear at a later date?

Occasionally, symptoms referable to the labyrinth may predominate and obscure other signs due to involvement of the pyramid. When it is considered that the route of invasion is frequently along the perilabyrinthine cells, it may be wondered that involvement of this structure is not encountered more often. Some patients, however, do present vertigo, vomiting and nystagmus, and when such signs are found care must be taken to rule out the possibility of a cerebellar lesion. That the process is not true labyrinthitis may be determined by hearing and vestibular tests, and the nystagmus will be found to vary both in intensity and duration from time to time. The labyrinthine symptoms may be so slight as to be almost overlooked, or they may be so severe as to overshadow the whole picture. The latter condition occurred in the following case:

CASE 3.—F. G., a boy aged 8 years, was admitted to the hospital March 30, 1939, with a history of having had a mastoidectomy on the left side on January 20. On March 13 his tonsils and adenoids were removed; this operation was followed by some postnasal hemorrhage. At the time of the mastoidectomy he had complained of temporal and occipital headache, and the temporal headache had continued at intervals. Two days before admission he had moderate pain in the epigastrium, accompanied by dizziness and vomiting. He was said to have had no fever with this attack.

On admission the patient was pale; the temperature was 98.4 F. and the pulse rate 94. He complained of dizziness when standing or sitting erect; this was relieved by lying down. Vomiting was induced by sitting up in bed, and there was some nystagmus to the right when he sat up, but this practically disappeared when he lay flat. When walking he had a staggering gait but went in no particular direction. He showed a well healed scar; the drum markings were not distinct, and there was a small amount of pus in the canal. The next day the boy complained of frontal headache, which he localized above and external to the left eye, and the vertigo persisted when he sat up. The temperature was 100.3 F. and the

white blood cell count 9,100. Examination of the spinal fluid showed it to be clear and under no increase of pressure; there were 7 cells. Roentgenographic examination of the petrous pyramid showed some "infiltration throughout with definite bone erosion along the whole superior surface from the superior semicircular canal to the tip." The following day the temperature rose to 101.3 F.; therefore, exploration of the pyramid was decided on.

On April 2 the operation on the pyramid was performed. The solid angle was removed down to the labyrinth. No fistula could be found. On elevation of the dura internal to the labyrinth, pus was encountered, after which the necrotic area of bone in the pyramid was located. A sufficiently large opening was made into the pyramid, and the cavity was drained with rubber. The vertigo rapidly disappeared, and the patient was discharged from the hospital on May 1.

COMMENT

Thus, 3 cases have been presented, in all of which the infectious process had ruptured through the cortex, forming a subdural abscess, and in all of which one or more of the typical signs were lacking. In 2 there was meningeal irritation, and in 1 there was not. In all the cases, however, the patient recovered.

The roentgenogram is a valuable aid to diagnosis. However, a positive finding does not indicate operative intervention; neither does a negative finding exclude it. Taylor [3] said: "Every change in the roentgenographic appearance of the petrous apex does not indicate a suppurative lesion of the apex." And Law [4] stated: "It is not possible to make a diagnosis of suppuration of the petrous pyramid from the information gained from the roentgenogram alone." It is only when taken in conjunction with the signs observed by other means that roentgenograms are of value. In all cases the roentgenogram should be taken from the three usual angles, a roentgenogram made from one angle acting as a check on that from another. Every effort should be made to determine if the process is advancing or declining. This can usually be done by a careful study from day to day, but even under the most watchful care signs of meningeal irritation may suddenly appear.

Of late there has been an effort on the part of some otologists to determine the exact site of the suppurative process in the pyramid. This may occasionally be done, but in by far the largest percentage of my cases there is no method by which this can be accurately determined.

Regarding the treatment of these conditions, it is agreed that no single procedure is suitable for all cases. In a case in which pyramid signs are present at the time of invasion of the middle ear, a simple mastoidectomy should be performed and a careful search for tracts made

3. Taylor, H. K.: Suppuration of Petrosal Pyramid: Roentgenologic Problems, Arch. Otolaryng. **18**:458 (Oct.) 1933.

4. Law, F. M.: Roentgen Examination of Mastoid Process, Am. J. Roentgenol. **31**:482 (April) 1934.

during this procedure. The result of this operation should be awaited before any further surgical intervention is attempted. Undoubtedly many patients recover spontaneously after a simple mastoidectomy. Two years ago I advocated an approach to the pyramid along the floor of the middle fossa and the avoidance of the radical mastoidectomy. By this approach one is able to reach the pyramid, drain the infected focus and have the patient recover with an intact middle ear and the preservation of good hearing. So far I have seen no reason to change this procedure, and feel that it is perfectly safe and not too difficult for common use. Objection has been made to this route on account of the likelihood of tearing the dura while carrying out the elevation or while draining the focus along the dura and of tearing the petrosal nerve. These objections are theoretic rather than practical, for no bad results have been observed in any case which could in any way be ascribed to this method of approach.

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PENETRATION OF PERINASAL TISSUES BY MERCUROCHROME

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AND
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Contrary to the usual assumption that the lymphatic vessels and the round window are the chief transmitters of solutions from the middle ear to the inner ear, Ross and Hamilton [1] demonstrated that mercurochrome placed in the middle ear would in a brief time penetrate the entire cochlea. This fact led us to believe that the introduction of mercurochrome into the nasal cavity would yield information regarding the absorption of material from the nasal mucosa and would reveal the sites to which the material passed and the routes it followed.

Mercurochrome was employed in this study because it is soluble, does not produce tissue necrosis or coagulation, stains tissues a brilliant red and can be precipitated in situ by mineral acids and retain its color, thus making it possible for its spread to be arrested at any desired moment.

Since little of the disease in the nose occurs in the first layer of cells, substances used to treat nasal infection must penetrate deeply into the tissues. Few chemical substances have been discovered that will do this; therefore, the possibility suggested itself that if mercurochrome were found to diffuse widely through the perinasal tissues, this or a similar drug might be used as an antiseptic agent. It was hoped that the mode of spread of this dye would reveal the path taken by therapeutic agents intended for deep tissues.

METHOD

Dogs weighing between 10 and 20 Kg. were used. The majority were anesthetized with injections of pentobarbital sodium, although some were anesthetized with ether administered through a tracheal cannula. The dogs were maintained in a supine or prone position with the nose held in the horizontal plane. Four cubic centimeters of an 8 per cent solution of mercurochrome was introduced

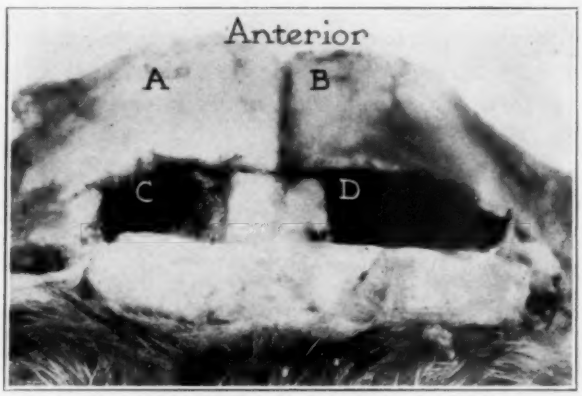
† Dr. Ross died on Dec. 21, 1938.

From the Departments of Otolaryngology, Physiology and Pharmacology, Northwestern University Medical School.

1. Ross, E. L., and Hamilton, J. W.: Penetration of the Inner Ear by Mercurochrome, Arch. Otolaryng. **29**:428-436 (March) 1939.

gently into the right nostril, where it was left for a period varying from a few minutes to seven hours. (Two hours gave the best results.)

Physiologic solution of sodium chloride containing 3 per cent hydrochloric acid was then perfused through the arteries of some of the dogs until they were dead. Perfusion with a solution of 5 per cent formaldehyde containing 3 per cent hydrochloric acid was then begun and continued until the return flow was clear. The animals were decapitated and either examined immediately or preserved in the same solution as that used for the second perfusion until they could be examined for location and depth of penetration of the mercurochrome. Two of the dogs were killed after two hours' exposure to the dye and were immediately decapitated; the heads were frozen almost instantaneously in a solution of absolute alcohol and dry ice at a temperature of —70 C. These heads were dissected in the frozen state. The remainder of the animals were examined immediately after the termination of the period allowed for the dye to remain in the nose. As controls, 8 dogs dead for several hours were treated in exactly the same manner as the normal dogs.



Photograph of a section made through the bregma of a dog to show penetration of mercurochrome into the bone. *A*, the left frontal bone, unstained; *B*, the right frontal bone, completely stained through by mercurochrome; *C*, the left frontal sinus (opened), unstained, and *D*, the right frontal sinus (opened), the walls deeply stained with mercurochrome.

By the following procedure the heads were examined for evidences of staining: A sagittal section was made just to the left of the midline so as to miss the nasal septum, and then a cross section was made just behind the ears. The brain was carefully teased out and examined, as was the dura mater; finally the underlying bone was also examined. The skin and soft tissues of the bregma were dissected away layer by layer, and the eyeball was removed. The frontal sinuses were opened, and, lastly, the nose was cut across in severe planes in order that the depth of the penetration of the mercurochrome might be visualized.

RESULTS

The mercurochrome introduced into the right nostril spread rapidly and completely throughout the nasal cavities until the entire mucous

membranes were stained a brilliant red and the right frontal sinuses were filled, except in 2 puppies with unopened sinuses and in 3 of the dead dogs.

In all of the dogs held in a prone position the penetration of the dye through the nasal mucosa and turbinates and into the underlying tissue, mainly bone and cartilage, was most marked from the region of the frontal sinus toward the cranial cavity. In all but 2 of these animals the mercurochrome was visible grossly within the cranial cavity, and in 4 the dura had been penetrated as well, the dye appearing on the tip of the frontal lobe in a sufficient quantity to stain it grossly.

The penetration of the mercurochrome into the perinasal tissues in the dogs held in the supine position was most marked through the frontal bone interiorly and superiorly from the frontal sinus, the overlying soft tissues of the bregma up to the skin being stained a definite pink. In 2 of these animals the mercurochrome had also passed completely into the cranial cavity, to stain grossly the dura and the tip of the right frontal lobe of the brain.

The right eye and orbital tissues of 11 of the dogs were removed to disclose a marked staining of the orbital side of the bony septum separating the orbit and the nasal cavity.

When the dogs dead from eight to thirty-six hours were examined after two hours of exposure to the intranasally administered dye, none of the mercurochrome could be found, grossly, beyond the nasal mucosa. Finally, a dog was killed one and one-half minutes after the dye was instilled and the mercurochrome left in the nose for a subsequent one and one-half hours. Examination revealed that there was no penetration of the stain through the nasal mucosa, for none of the underlying tissues showed the slightest discoloration.

The observations are recorded in detail in the table, and the photograph shows the penetration of mercurochrome into the bone.

COMMENT

The olfactory mucous membrane is highly absorptive for many types of particulate matter and for many viruses. Faber [2] not only confirmed the observations of Clark [3] pertaining to this, but correlated them with earlier retrograde studies, when he demonstrated that the definite anatomic channels of nasal absorption are the same as those by which

2. Faber, W. M.: Nasal Mucosa and Subarachnoid Space, Am. J. Anat. **62**:121-148 (Nov.) 1937.

3. Clark, W. E.: Report to the Committee on Vaccination on an Anatomical Investigation into the Routes by Which Infection May Pass from the Nasal Cavity into the Brain, Reports on Public Health and Medical Subjects, no. 54, Ministry of Health, London, His Majesty's Stationery Office, 1929.

Observations of Staining of the Perinasal Tissues

Number of Animal	Condition	Hours of Stain	Type of Anesthesia	Position	Examination	Right Frontal Sinus	Anterior Wall of Brain Case	Underlying Dura	Frontal Lobe of Brain	Olfactory Structures	Frontal Bone, Antero-superior	Soft Tissue of Bregma	Medial Wall, Right Orbit
34	Dead 24± hours	2	.....	Prone	Immediate section	.....	.....	.....	.....	.....	.....	.....	.....
39	Dead 10± hours	2	.....	Prone	Immediate section	Filled by dye	.....	.....	.....	.....	.....	.....	.....
40	Dead 10± hours	2	.....	Prone	Immediate section	Filled by dye	.....	.....	.....	.....	.....	.....	.....
41	Dead 10± hours	2	.....	Prone	Immediate section	Filled by dye	.....	.....	.....	.....	.....	.....	.....
35	Dead 36± hours	2	.....	Supine	Immediate section	Filled by dye	.....	.....	.....	.....	.....	.....	.....
36	Dead 10± hours	2	.....	Supine	Immediate section	Filled by dye	.....	.....	.....	.....	.....	.....	.....
37	Dead 10± hours	2	.....	Supine	Immediate section	Filled by dye	.....	.....	.....	.....	.....	.....	.....
38	Dead 10± hours	2	.....	Supine	Immediate section	.....	.....	.....	.....	.....	.....	.....	.....
7	Lived ½ min. after insertion of dye	1½	Pentobarbital sodium	Prone	Perfusion; section later	.....	.....	.....	.....	.....	.....	.....	.....
12	Alive	¼	Pentobarbital sodium	Prone	Perfusion; immediate section	.....	Penetration, 1.0 mm.	.....	.....	.....	.....	.....	.....
2	Alive	1	Pentobarbital sodium	Prone	Perfusion; section later	Filled by dye	Stained through	Stained	Right tip stained	.....	.....	.....	Not examined
9	Alive	1¼	Ether	Supine	Immediate section	Filled by dye	.....	.....	.....	.....	Stained through	Stained	Not examined
10	Alive	1¼	Ether	Supine	Immediate section	Filled by dye	Stained through	Stained	.....	.....	Stained through	Stained	Stained through

32	Alive	1¼	Pentobarbital sodium	Prone	Frozen; section later	Filled by dye	Stained through	.....	.....	.....	Stained through	Stained	Stained through
8	Alive	2	Pentobarbital sodium	Prone	Perfusion; section later	Filled by dye	Stained through	Stained	Right tip stained	.....	.....	.....	Not examined
13	Alive	2	Pentobarbital sodium	Prone	Perfusion; section later	Unopened (pup)	Penetration, 2.5 mm.	.....	.....	.....	.....	.....	Stained through
14	Alive	2	Pentobarbital sodium	Prone	Perfusion; section later	Unopened (pup)	Stained through	.....	.....	.....	.....	.....	Stained through
15	Alive	2	Pentobarbital sodium	Prone	Perfusion; section later	Filled by dye	Stained through	Stained	Right under surface stained	.....	.....	.....	Stained through
33	Alive	2	Pentobarbital sodium	Supine	Perfusion; section later	Filled by dye	Stained through	Stained	Right tip stained	Cribriform plate stained	.....	.....	Stained through
11	Alive	2	Pentobarbital sodium	Supine	Perfusion; immediate section	Filled by dye	.....	.....	.....	.....	Stained through	Stained	Not examined
29	Alive	2	Ether	Supine	Immediate section	Filled by dye	.....	.....	.....	.....	Stained through	Stained	Not examined
30	Alive	2	Ether	Supine	Immediate section	Filled by dye	Stained through	Stained	Right tip stained	.....	Stained to periosteum	.....	Stained through
42	Alive	2	Pentobarbital sodium	Supine	Immediate section	Filled by dye	.....	.....	.....	.....	Stained through	.....	Stained through
43	Alive	2	Pentobarbital sodium	Supine	Immediate section	Filled by dye	.....	.....	.....	.....	Stained through	Stained	Stained through
31	Alive	3½	Pentobarbital sodium + ether	Supine	Immediate section	Filled by dye	.....	.....	.....	.....	Stained through	Stained	Stained through
6	Alive	7	Pentobarbital sodium	Prone	Perfusion; section later	Filled by dye	Stained through	Stained	.....	.....	.....	.....	Stained through

materials placed in the subarachnoid space gain exit from the cranial cavity. Aqueous solutions have been proved to pass both along olfactory nerve sheaths and to cervical lymphatics, while the viruses of poliomyelitis, herpes simplex, yellow fever, vesicular stomatitis and louping ill are neurotropic and enter the central nervous system by growth either along the axis cylinders or in the perineural spaces of the olfactory nerves, and presumably not through lymphatic channels.

Our results with mercurochrome indicate another route or mechanism by which certain materials in contact with the mucosa of the upper respiratory tract may penetrate the tissues. It was shown by our experiment that mercurochrome directly penetrates and diffuses through the nasal mucosa and surrounding tissues, even into the cranial cavity. It apparently follows no definite path, but diffuses in all directions from the frontal sinus and nasal antrum into the soft tissues, to the dura mater and even onto the tips of the frontal poles of the brain itself. This can occur in a surprisingly short time. The methods of examination used serve to rule out postmortem spread. In no case had the stain penetrated the cribriform plate, and it was not found grossly along the olfactory nerves or in the region of the olfactory bulbs. Comparable staining did not occur in the dead animals. This indicates that the movement of tissue fluids is essential for the penetration. Since the area of spread is so limited and uniformly stained, it would seem that the spread of mercurochrome is not confined to lymphatic and blood vessels but occurs through the intervening tissue spaces, as suggested by Weed.[4] Although the mercurochrome appeared to spread concentrically from the nasal cavity, closer inspection revealed lightly stained areas rich in lymphatic channels, from which the dye apparently drained away, and other areas deeply stained where anatomically lymphatic capillaries and loose areolar tissue were at a minimum.

Just as we observed mercurochrome to penetrate the nasal mucosa but not the cribriform plate, Yoffey and Drinker[5] found that Evans' blue dye (T-1824) and trypan blue passed into the cervical lymphatics but did not penetrate the cribriform plate. We agree with their explanation of this deviation from the results given in earlier reports. It is quite tenable that in a small animal, such as the mouse, in which the cribriform plate is an exceedingly tenuous structure, this plate might permit the passage of dyes to which the thicker one of larger animals would be more resistant.

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4. Weed, L. H.: Studies on Cerebrospinal Fluid, J. M. Research **31**:51-118 (Sept.) 1914; The Cerebrospinal Fluid, Physiol. Rev. **2**:171-203 (April) 1922.

5. Yoffey, J. M., and Drinker, C. K.: The Lymphatic Pathway from the Nose and Pharynx: Absorption of Dyes, J. Exper. Med. **68**:629-640 (Oct.) 1938.

SUMMARY AND CONCLUSIONS

Mercurochrome introduced into the nose and retained there during life will pass through the mucosa of the nose and sinuses, through the turbinates, through the bony walls of the frontal sinus anteriorly, superiorly and posteriorly and even through the dura, to discolor the cortex of the brain in this area in much less than two hours. This transmission is mostly by diffusion, as is known by the rather general concentric spread throughout all of the perinasal tissues, though the process occurs to an appreciable extent only in the living animal.

INDURATIVE OR MYALGIC HEADACHE

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Cabot [1] in his book on differential diagnosis quotes Edinger as follows:

> This probably the most frequent form of headache—seems to be unknown to the majority of physicians although it has been described in textbooks for decades. Among the cases I have seen, which far exceed one hundred, few have been properly diagnosticated and treated.

This condition was first pointed out to me in Berlin, Germany, by Halle in 1925. Since that time I have seen hundreds of patients with indurative headache, many of whom have been referred to me with incorrect diagnoses. The most frequent mistake made by general practitioners is to confuse the pain in the head caused by myalgia of the muscles of the neck with disease of the accessory sinuses, otitis media and mastoiditis. In several cases a diagnosis of meningitis was made. One woman attempted to commit suicide because of severe headaches, which she had been repeatedly told were due merely to nervousness. I have found in conversing with my colleagues that many are not cognizant of this condition; this is not surprising, since little has appeared in English on indurative or myalgic headache. This is especially true in regard to the otolaryngologic literature. The first reference that I can find in the otolaryngologic literature relative to myalgia is that of Halle [2] in 1911. The only contribution on this subject in the American otolaryngologic literature is by Mithoefer.[3]

The term indurative headache expresses an attempt to characterize the malady without committing oneself to any theory relative to its cause or pathologic mechanism. The name is derived from the presence of indurated areas or nodules in the cervical muscles. Since "indurative headache" is rather ambiguous, I have added the name myalgic to show the origin of the condition more clearly. I may add that in a given case there may be spasm of one or more of the cervical muscles, either with or without the appearance of nodules. The pain in the head, which I shall describe in detail later, is a result of the aforementioned nodular

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1. Cabot, R. C.: Differential Diagnosis, Philadelphia, W. B. Saunders Company, 1915, p. 35.
2. Halle, M.: Monatschr. f. Ohrenh. **45**:768, 1911.
3. Mithoefer, W.: Tr. Am. Laryng., Rhin. & Otol. Soc. **40**:284, 1934.

or spastic condition of the muscles, which occurs almost universally. In the majority of persons, however, it is transient and mild, and they make little or no complaint. In cases of extreme pain on the other hand, a patient may be so afflicted as to become a veritable invalid. In this paper reference will be made only to headaches caused by noninflammatory processes in the cervical muscles.

The cause of this condition has not as yet been definitely established. I am of the opinion that the following predisposing factors play a part. The patient's history usually presents evidence of vasomotor imbalance, and frequently he is found to have some form of allergy. Many patients are hypersensitive, are under high nervous tension or are psychoneurotic. It is also probable that many have metabolic or endocrine disturbances, while others suffer from various forms of chronic arthritis. Occupation and environment are important factors; i. e., persons who are obliged to hold their heads in fixed positions for long periods are more subject to this complaint. Myalgia occurs almost exclusively in the postural muscles.

Indurative headaches may occur at any age but occur more often in adult life. I have seen patients with myalgia at every age, even in early infancy, and in every walk of life. My own experience, both personal and in the observation of my patients, leads me to believe that all these patients are sensitive to cold and that the exciting factor in the production of myalgia seems to be exposure to drafts and cold. There is of course a great variation in the degree of susceptibility to drafts and exposure in various persons and in the same person at different times. In some patients, the slightest movement of air, even within a closed room, may cause symptoms, while others can tolerate considerable exposure.

Most patients find it impossible to be outdoors without a head covering and cannot sleep in drafty rooms or ride in automobiles with the windows open. Chilling of the body or of the feet, even though the head and neck are fully protected, often brings on an attack. Headache frequently follows shampooing of the hair, especially if an electric dryer is used. Edinger [4] stated that several patients with myalgia have been relieved by wearing a wig. Exposure to the drafts of an air-conditioned room or train is frequently followed by headache. There is little difference in the number of cases observed during summer or winter, but seasonal changes, variations in atmospheric pressure and especially changes from a dry to a damp climate definitely increase the number and severity of attacks in susceptible persons.

4. Edinger, L.: Diseases of the Nervous System, edited by A. Church, New York, D. Appleton and Company, 1909, p. 863.

All patients are more susceptible during fatigue, after severe exertion or during focal infections. Many patients state that their symptoms were first noticed after an acute illness, especially some infectious disease. It seems possible that they were sensitized to cold during the infectious process. Finally, a number of authors have stated the belief that myalgia is an allergic manifestation.

The headache caused by nodular or spastic myalgia may or may not be accompanied by conscious pain or soreness in the cervical muscles. Most frequently the patient is unaware of any discomfort until his attention is called to it by palpation of these muscles. When pain in the cervical muscles is referred to by the patient, it is always in a minor way. As a matter of fact, the patient rarely connects the pain in his head with the spasm in his cervical muscles. Again, there are many persons who constantly have stiffness and pain in the cervical muscles but who never have concomitant headaches. The headache may be localized in the occiput, in the supraorbital, temporal area, in the frontal region, over the maxillary sinuses or behind the eyes, or it may be referred to the teeth, ears or throat. In other cases the pain may be diffuse, either manifesting itself as hemicrania or affecting the entire head. The pain varies greatly in degree and character in different patients and in the same patient at different times. It may be mild and dull or sharp and lancinating; at times there may be a constant severe throbbing pain with intermittent lancinating exacerbations. The pain may be so slight as to be hardly noticeable or so severe as to be almost unbearable.

In cases of chronic pain, in either mild or moderately severe attacks, the pain may occur repeatedly in the same area. On the other hand, it may move from place to place even within a short period. The pain in the head most frequently begins in the occipital region, or, to be exact, in the region of insertion of the cervical muscles to the occipital bone, and may remain in this location during an entire attack. On the other hand, it may radiate to the temporal or the frontal region or even to the upper teeth. When this occurs it resembles exactly the sensation experienced in the teeth in disease of the maxillary sinus. In other cases the entire pain may be localized deep in one ear, or both ears. As a rule it is much less severe when it remains localized in the occipital region. In some patients the pain extends from the trapezius muscle down into the arm and hand.

Pain in the head may begin at any time of the day or night. Frequently it comes on during the night as a result of an unnatural position of the head on the pillow, of sleeping in a draft or of a combination of these conditions. It may become so severe that the patient finds it impossible to remain in bed. He is more comfortable in the upright position, especially when the head is inclined forward and supported by

his hands. In other instances the attack is not of sufficient severity to prevent sleep but is first noticed after arising. The headache in these patients usually improves after exercise and may disappear in the morning or afternoon, thus giving the impression of a sinus headache.

Pain in the frontal region frequently radiates to the bridge of the nose and to the orbit, especially on its median wall, or it may be referred to the depth of the orbit. At times the pain is so severe that it is impossible for the patient to move his head or mimic muscles or to swallow. An interesting patient with this type of pain came to my office with her head drawn completely to one side and with her chin in contact with her clavicle. Her complaint was that she had a sore throat and could not swallow. It was almost impossible for her to open her mouth owing to the pain in her throat. She was found to have not a sore throat but a painful area in the cervical muscles. Almost immediately after procaine hydrochloride was injected into this area the patient obtained complete and permanent relief.

In some cases of mild or beginning indurative headache an attack may last for three or four days and may not return for months or years. In others the attacks occur more and more frequently, until finally the patient may become a veritable recluse, each exposure to fresh air or to a draft bringing on an attack. I have termed this phase status myalgicus. In some instances the onset is sudden, following an exposure to cold. In the majority of cases, on the other hand, there is an interval between exposure to cold and onset of the pain. Freund [5] stated that during the war, when soldiers were exposed to prolonged periods of cold and dampness, the onset of the myalgia was slow and the condition usually did not reach its peak until from two to four weeks had elapsed. Patients frequently state that they have no pain when out in the cold but sense its onset soon after entering a warm room.

In addition to the pain in the head there is frequently a feeling of nausea, a loss of desire for food, and giddiness, or vertigo, which is usually increased by stooping. Some writers have also reported tinnitus (Halle [2]). During the attack, especially if the pain is frontal, there are frequently drooping of the eyelids, difficulty in thinking, irritability, insomnia and fatigue. When the pain becomes more or less general there may arise a feeling of contracture of the pharyngeal muscles, i. e., a "choking sensation," or even severe cramplike pain in the throat. Wodak [6] referred to a case in which there was severe pain in swallowing, which lasted over a month. A few cases of myalgia with severe pain in the facial muscles have been reported.

The objective findings are the muscular nodes, muscular spasms and painful areas in the cervical muscles. In this respect also a great varia-

5. Freund, E.: Acta rheumatol. **8**:32 (Aug.) 1936.
6. Wodak, E.: Arch. f. Ohren-, Nasen- u. Kehlkopfh. **109**:63, 1922.

tion occurs in different patients or in the same patient at different times as to the sensitivity of the muscles to palpation. The degree of sensitivity, however, is not in any way related to the severity of the concomitant headache. Patients with myalgic headaches almost always have tenderness in one or more of the cervical muscles. There are patients who react so strongly to palpation of these muscles that one is inclined to think that they exaggerate. Others acknowledge only with reluctance that a muscle is sore. Comparative palpation of other muscles should always be made.

The method of examining the muscles is important. The nodules are detected by using the tips of the first three fingers, palpating first the trapezius and the sternocleidomastoid muscles and then, with greater pressure, the deeper ribbon muscles of the neck. The search must be systematic. The finger tips exert pressure on the muscles by using a circular movement. Spasms are detected by picking up the edge of the muscle and rolling it between the thumb and the first and second fingers.

At times the sternocleidomastoid muscle stands out so that it is plainly visible. At other times the edge of the trapezius muscle may present a raised border. Under these conditions the edge of the muscle feels indurated. This feeling of induration is also frequently present when there is no apparent thickening. Nodules may be found in any of the cervical muscles and may be single or multiple. The finding of nodules in the orbicularis muscle is also rather frequently mentioned. In many cases there is an indurated area at the insertion of the cervical muscles. It must be emphasized that the nodules are not usually the seat of the most severe pain. Pain arising from muscles is always diffuse and often referred and is associated with referred tenderness of the deep structures. Pressure often reveals sensitive areas pressure on which reproduces or accentuates the pain. The skin over the nodules and over the muscle insertions—in fact, the entire scalp—may become hypersensitive to touch, especially after a severe attack of pain in the head.

Since myalgia is responsible for so much headache and also because it so often is confused with other conditions that otolaryngologists are called on to treat, it is essential that indurative headache be ruled out whenever a patient complains of headache. Myalgic pain is most frequently mistaken for that caused by sinusitis. Many of the patients who came to me with a self-made diagnosis of sinal pain were really suffering from myalgia. Since the pains occur both over the frontal sinuses and over the antrums and since there are often discomfort and a feeling of elongation of the upper teeth, it is easy to see how these conditions can be confused.

The pain resulting from myalgia is also commonly confused with that caused by otitis media and mastoiditis. This error may occur more easily if the myalgia is accompanied by giddiness, tinnitus and nausea. After a simple mastoid operation 1 of my patients had a severe retro-orbital pain resembling exactly the pain produced by petrositis. A number of cases have been reported in the literature in which operations on the mastoid and other surgical procedures were performed because of the severe pain which was referred to the structures in this region by myalgia.[7] A number of patients have been referred to me for surgical intervention when the cause of the pain was likewise myalgia. Pain in the throat and neck resulting from this condition may be confused with various infections in the throat and neck. A diagnosis can frequently be made from a careful history.

It is usually not difficult to differentiate indurative headache from the various conditions that produce similar symptoms. It must be remembered, however, that it is frequently associated with other conditions and diseases, such as sinusitis, suppurative otitis media, chronic arthritis and hypertension. Under these circumstances the various symptoms and findings must be carefully considered before diagnosis is made. Glaser,[8] in a paper on atypical facial neuralgia, submitted a comprehensive outline on the differential diagnosis of headaches. This outline, somewhat modified, I am presenting in this paper.

Atypical facial neuralgia, according to Glaser, "has many things in common with true migraine, but in addition it has some points in common with neuralgias of the cranial nerves, particularly with trigeminal neuralgia." Glaser's classification (modified) of atypical facial neuralgia is as follows:

I. Primary atypical facial neuralgia. For this no cause can be ascertained, and treatment must be directed toward the blockage of sensory pathways. The diagnosis is reached only by a process of elimination.

II. Secondary atypical neuralgia due to various clinical entities.

1. Sphenopalatine and vidian neuralgia.
2. Postherpetic trigeminal neuralgia
3. Trigeminal ghosts
4. Trigeminal ghosts with lingual spasm
5. Temporomandibular joint syndrome
6. Autonomic faciocephalalgia
7. Painful convulsive tic
8. Senile neuralgia

7. Halle.[2] Mithoefer.[3]

8. Glaser, M. A.: Atypical Facial Neuralgia: Diagnosis, Cause and Treatment, Arch. Int. Med. **65**:340 (Feb.) 1940.

III. Atypical facial neuralgia produced by systemic diseases.
   1. Allergy
   2. Endocrine disturbances
   3. Psychoneurosis

IV. Atypical facial neuralgias due to lesions of the head, chest and abdomen.
   1. Infections about the head
      (*a*) Mastoiditis
      (*b*) Sinus infections
      (*c*) Deep-seated facial abscess
   2. Tumors of the neck and head
   3. Intracranial lesions
   4. Dental sepsis
   5. Deviations and spurs of the nasal septum
   6. Ocular lesions
   7. Lesions of the chest
   8. Pathologic conditions in the abdomen and pelvis

It is not possible for me to refer to each of the conditions which he has mentioned. For this I refer the reader to his article. I shall, however, outline some of the salient features of several of the less known conditions which he has described.

The pain of primary atypical facial neuralgias may be located in the same areas as that caused by myalgia of the cervical muscles but differs from it in that it is usually continuous. In about 50 per cent of the reported cases it is accompanied by phenomena referable to the sympathetic nervous system. These phenomena consist chiefly of ocular disturbances, such as lacrimation, edema, corneal injection, inequality of the pupils, blurred vision, photophobia and enophthalmus. In addition, some of the following symptoms are frequently present: nausea, vomiting, flushing of the face, nasal discharge, perspiration, salivation, puffiness of the face, a feeling of warmth, ringing of the ears, soreness over the temporal artery and chills. The condition is found chiefly in females and is bilateral in 33 per cent of the cases. In Glaser's series of 245 patients with atypical neuralgia, less than 10 per cent were relieved of their pain.

Concerning autonomic facioencephalalgia, Brickner and Riley[9] advanced the opinion that there are two types of atypical migraine, one influenced by epinephrine (and presumably dependent on localized hypofunction of the sympathetic nerves) and the other influenced by the supposed antagonist of epinephrine, ergotamine tartrate (and presumably dependent on localized hyperfunction of the sympathetic nervous system).

9. Brickner, R. M., and Riley, H. A.: Bull. Neurol. Inst. New York **4**:442 (Dec.) 1935.

Painful convulsive tic is a condition in which the patient suffers with severe facial spasms associated with considerable pain. The condition is rare and is not relieved by operation on the gasserian ganglion.

Finally, I shall mention the so-called vasomotor headache, which was described by Cabot in 1915; this has recently been brought to the attention of otolaryngologists by Horton,[10] of Rochester, Minn., and called by him erythromelalgia. The patients suffer with a unilateral headache of sudden onset. There are flushing of the face on the involved side and an increase in the surface temperature of from 1 to 3 degrees C. (1.8 to 5.4 degrees F.) on the affected side. The headaches are caused by cerebral vasodilatation and are relieved by administration of vasoconstricting substances, such as ephedrine. Recurrences have been prevented by the use of histamine given subcutaneously.

PATHOGENESIS

First I shall cite various theories that have been advanced from time to time as to the cause of the localized muscular spasms or nodules which occur in indurative headache and the pain which accompanies them.

1. Ditmar [11] expressed the opinion that they are due to inflammatory reaction.

2. Volta [12] stated that the cause is a pathologic process and that it is difficult to trace the exact line of demarcation between myalgia and myositis. He stated that the nodules are caused by changes in the interstitial lymph and by increased tone of the muscle fibers resulting from hyperstimulation of the motor nerve fibers.

3. A Mueller [13] asserted that the nodules are due to increased tension in the muscle fibers.

4. Schulhof and Ruhman [14] stated the opinion that the nodules are due to disturbance in the capillary circulation caused by abnormal vessel reflexes.

5. According to Deller and Geckelhorn,[15] there is a change in the electrolytic potential between the tissues and the tissue fluids.

6. Shade and Lange [16] and others expressed the belief that the nodules are caused by changes in the muscle colloids.

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10. Horton, B. T.; MacLean, A. R., and Craig, W. M.: Proc. Staff Meet., Mayo Clin. **14**:257 (April 26) 1939.
11. Ditmar, O.: Med. Welt **6**:849 (June 11) 1932.
12. Cirera Volta, R.: Acta rheumatol. **8**:43 (Aug.) 1936.
13. Mueller, A.: Der musculäre Kopfschmerz, Leipzig, F. C. W. Vogel, 1911.
14. Schulhof and Ruhman, cited by Freund.[5]
15. Deller and Geckelhorn, cited by Freund.[5]
16. Shade and Lange, cited by Freund.[5]

7. Goldsheider, Foster, Lilyan and Mayle [17] came to the conclusion that cold interferes with the action of the osmotic active ions of the colloids. They said that it is possible that this process of changed ionic concentration is the cause of alteration in the excitability of the muscle as well as of the myalgic pain. They also stated that muscular fatigue may cause increased permeability of the vessels in the muscles, thus producing myalgia. The aforementioned process is characterized chemically by an increase of lactic acid, ammonia and phosphoric acid and a reduced ability to rebuild hexosephosphate (lactacidogen). The accumulation of these products producing muscle spasms will further impede the normal metabolic processes.

8. Quincke and Eppinger [18] stated that the condition is similar to that associated with urticaria, i. e., due to an increased permeability of the capillaries. They expressed the opinion that there is a transudation of serum into the tissue spaces, hindering the normal exchange of nutriments. This results in anoxemia and lack of elimination of the products of metabolism (uric acid, etc.).

9. Gudzent, Curshmann and Freund [19] and others asserted that the nodules are due to allergy, the pain and rigidity being caused by infiltration.

My experience with several hundred cases of nodular myalgia makes me conclude that none of these theories satisfactorily explains the pathogenesis of this condition. First, I think that it has been shown that the process is not infectious; e. g., Schmidt [20] and others excised painful nodules from several patients with myalgia. Sections failed to reveal any pathologic changes. In addition, I add this evidence: The nodules can frequently be made to disappear within a few minutes by injecting procaine hydrochloride into them. Finally, nodules disappear spontaneously and others reappear without leaving apparent permanent changes in the muscle even in cases in which the process has been present for years. There is undoubtedly an increase in the tension of the involved muscle fibers, and also there are undoubtedly abnormal vessel reflexes, but these are merely incidental. The same may also be said of the theories relative to changes in the muscle colloids. The question of allergy as a causative factor will be presented in more detail later in the paper.

17. Goldsheider, Foster, Lilyan and Mayle, cited by Freund.[5]

18. Quincke and Eppinger, cited by Freund.[5]

19. Gudzent, Curshmann and Freund, cited by Freund.[5]

20. Schmidt, A.: Der Muskelrheumatismus, Bonn, Verlag A. Marcus & E. Weber, 1922.

Before attempting to find a better solution to this problem, I believe that it is necessary to take into account the following personal observations:

1. All of my patients were sensitive to cold, especially drafts.

2. Most of them presented symptoms of vasomotor instability.

3. Frequently, especially during a severe attack of indurative headache, the patient presented a difference in temperature on each side of the neck. Not having a thermocouple, I should judge the difference to be as much as 2 to 3 degrees (F.), the colder side being always that of the greater pain.

I shall now present a number of studies of the literature made in the attempt to solve the problem of the pathogenesis of this condition. My first was an attempt to determine what reaction took place in the vessels of the cervical muscles of a myalgic person exposed to draft. The work of Lewis [21] on the skin has shown that the function of blood vessels there is primarily to assist in regulating the body temperature. The vessels of this organ react in a characteristic manner to stimulation, i. e., after mild scratching, stretching or freezing or stimulation of the skin with a galvanic current a localizing blanching (white line) occurs, which is due to a contraction of the walls of the smaller vessel and is independent of nervous reflex. This action is similar to that of injected epinephrine or pituitary extract. After more vigorous stimulation there arises around the blanched area the so-called flare, or area of hyperemia, which is caused by an axon reflex. This is a local reflex, the stimulating impulse traveling up one loop of a sensory nerve and down the other. In other words, the afferent tract is sensory, and the efferent channel begins as a collateral branch of a fiber of the same system. This action requires no interpolated nerve cell in its path. Finally a wheal is formed in the area which was formerly red. There follows an exudate of fluid from the vessel that participated in the local reaction.

The original stimulus is not itself the cause of the formation of the flare but acts by damaging the tissue, liberating a diffusible substance in the skin. This in turn provokes the reflex vascular reaction by its direct effect on the vessel walls, causing their dilation. The substance released by the injured vessels Lewis termed the H substance, which is closely akin to histamine. It has also been shown that any form of stimulus known to produce painful sensations will also elicit this reflex.

Lewis further stated: "There is no doubt that similar reactions occur in the deeper lying tissues, although these reactions have not been explored in any detail it is clearly probable that similar agencies are here at work and that the skin does not stand alone in exhibiting the phenomena described."

21. Lewis, T.: The Blood Vessels of the Human Skin and Their Responses, London, Shaw & Sons, Ltd., 1927.

These dermal reactions following irritation are similar to those reported by several authors as occurring in cases of allergy, and, since a number of European writers on indurative headache have stated that they think the condition due to allergy, this subject was next investigated. If allergy is the cause of this condition it must of necessity be a physical allergy. Duke [22] stated:

> Reactions caused by sensitiveness to heat, effort or to cold vary in intensity, in the quickness in which they appear and in location. In some individuals, the reactions are chiefly constitutional and manifest themselves as syncope, effort syndrome, heat prostration, or shock. In others, where the reaction is referable chiefly to the cooling surface, the pathological manifestations may be non-infectious coryza, asthma, cough, pruritus, erythema, urticaria, angioneurotic edema or eczema. In those in whom the reactions are referable chiefly to internal organs the response may be tachycardia, anginal pains, nausea, vomiting, diarrhea, convulsions, coma, headache or other symptoms referable to one or several structures. Again physical allergy may be characterized either by local manifestations more or less confined to the area affected by the physical agent or by local and widespread manifestations in constitutional reactions and eosinophilia or in distant structures only. Restoration to a normal state could be accomplished by exposure to an agent the reverse of the one which caused the reaction.

Most authors agree that (1) it has been fairly well established that histamine is a normal constituent of living cells; (2) whenever living cells are injured histamine is released into the blood or surrounding tissues, and (3) the antigen-antibody reaction in sensitized animals is capable of acting as a stimulus to cause release of histamine from living cells. That histamine is liberated from the cells in cases of physical allergy has also been shown by Roth and Horton.[23] These investigators, as well as others, have been able to desensitize patients with physical allergy by hypodermic injections of histamine and also by the use of histaminase, which is an enzyme derived from the intestinal mucosa and capable of neutralizing histamine.

From a theoretic standpoint, physical allergy would seem a complete answer to the problem were it not for the following facts: In some cases of the most severe nodular headache the neck on the involved side is colder than on the unaffected side. Histaminase, although it has been of slight temporary value, neither prevented nor cured an attack of indurative headache when used by me. Efforts to desensitize patients with histamine and with ice failed. Finally, in a series of unselected cases, examination of the blood revealed no eosinophilia except in a few patients who were definitely allergic.

I shall now advance a theory relative to the pathogenesis of myalgia, which takes into consideration the following factors that I have observed

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22. Duke, W.: J. Allergy **3**:257 (March) 1932.

23. Roth, G. N., and Horton, B. T.: Proc. Staff Meet., Mayo Clin. **12**:7 (Jan. 6) 1937.

in indurative headache: (1) hypersensitivity to cold, (2) vasomotor instability and (3) in certain cases a local reduction of temperature on the side of the neck from which the pain arises.

It is an established fact that the vessels of the skin contract when exposed to cold in order to conserve the body heat. That a simultaneous contraction of the blood vessels of the kidneys takes place has been shown by Muller, Peterson and Rieden.[24] Although I cannot find any definite proof in the literature, it seems likely that a similar contraction may take place in the vessels which supply the cervical muscles. Under normal conditions, after exposure of the skin to cold a contraction (vasconstriction) of its vessels takes place, resulting in an increase in blood pressure and general body metabolism. The heat center is stimulated; body temperature is increased, and in due time the peripheral vessels relax, vasodilation takes place and a physiologic cycle has been completed. This cycle may not be completed in persons who suffer with myalgia; the first phase, that of vasocontraction, takes place much more easily than in persons not subject to this condition, owing to the lowered threshold of the sympathetic system. Sympathetic stimulation produces in this person a hypersecretion of some metabolite, similar to sympathin, which prevents the second phase of the cycle, namely, vasodilation. Prolonged vasocontraction produces ischemia and anoxemia of the involved muscle, prevents normal muscle metabolism and is characterized chemically by accumulation of the waste products of metabolism and possibly by changes in the muscle colloids.

The end result of this process is a localized (nodular) or generalized contraction of the involved muscle, producing pain. The pain in turn is capable of stimulating further the vasoconstrictor fibers of the sympathetic system, thus producing a vicious cycle which may continue until the causes which produce the stimuli have been temporarily removed or until stimulation of the parasympathetic (vasodilator) fibers becomes sufficient to produce vasodilatation.

The next problem that attracted my attention was that of explaining the characteristics and origin of myalgic pain. First I shall take up the characteristics of muscular pain. Lewis[25] showed that the quality of pain provoked from somatic structures depends more on the structure stimulated than on the nature of the stimulus. To provoke pain he injected small quantities of various substances into such structures as muscle, tendon and periosteum and found that in each structure the procedure gave rise to pain which was characteristic of the structure. In particular, he found that the pain produced by injecting such substances

24. Muller, E. F.; Peterson, V. F., and Rieden, W.: Med. Welt **12**:1731 (Dec. 3) 1938.

25. Lewis, T.: Brit. M. J. **1**:321 (Feb. 12) 1938.

into muscle is identical with that resulting from muscular contraction under ischemic conditions and therefore represents true muscle pain.

Lewis interested Kellgren [26] in the study of muscle pain, and in 1938 the latter author published two papers describing the results of his research. In a general way he found that:

1. Fascia and sheath give sharply localized pain, while muscles give diffuse pain which is referred.

2. The diffuse pain from a given muscle is always distributed within certain regions, though the distribution within these limits varies from individual to individual and according to the part of the muscle stimulated.

3. Pain from muscle may be confused with pain arising from other deep structures, such as joints.

4. The distribution of the diffuse pain from muscle appears to follow a spinal segmental pattern. This pattern differs from that of segmental innervation of the skin.

5. Referred pain from muscle is associated with referred tenderness of the deep structures.

In another paper, published at about the same time, he stated:

"Pain arising from muscle is always diffuse and is often referred and is associated with referred tenderness of the deep structures. Pressure on these spots sometimes reproduces the patient's pain."

To trace the origin of the pain is much more difficult. In fact, the problem is far from being solved. Little is known about the pain-afferent nerve endings in muscle. The same may be said of the region of the brain in which the impulses from the pain sense organs end. It is thought that it is mainly in the thalamus. Lewis [25] suggested that the factor that produces pain in an ischemic muscle is a chemical substance which normally passes into the tissue spaces and is removed by the blood stream. This substance accumulates in conditions of ischemia and so gives rise to pain. The pain of indurative headache is not of the acute type associated with neuralgias of the peripheral nerves, such as trigeminal and glossopharyngeal neuralgia, but, as Glaser [3] pointed out, is more like the pain associated with vascular disease. It is a well known fact that vessels are sensitive to pain; this is demonstrated by such conditions as Raynaud's disease, angina pectoris and embolism. Glaser, in his article on atypical neuralgia, showed that the distribution of the pain in this condition is in a circular area within the distribution of the vascular supply of the face and head.

With this distribution of pain, the question naturally arises whether the sympathetic system is responsible for the conduction of the painful sensations which arise in cases of indurative headache. There is still a considerable difference of opinion as to whether the sympathetic system is purely motor in function. At present the consensus seems to be that

26. Kellgren, J. H.: Clin. Sc. **3**:175 (April) 1938; Brit. M. J. **1**:325 (Feb. 12) 1938.

it is. The distribution of pain in indurative headache may, on the other hand, be otherwise explained. Hunt's [27] views on the clinical aspects of neuralgia may well be applied to this condition. He wrote:

> Neuralgia while originally a painful affection of a single nerve or filament, tends gradually to diffuse itself in the distribution of the whole ganglionic system. In very severe neuralgia the pain may radiate and even pass beyond the boundaries of the affected ganglion distribution into other areas: whether this takes place by reason of peripheral anastomoses with neighboring systems or through central connections with neighboring systems or through central connections in the sensory nuclei of the brain stem is not known.

Why is it that myalgia, at least as far as I know, occurs only in the muscles of the neck and back? In searching for the answer one discovers that these muscles are postural muscles and as such vary physiologically from the muscles which execute movement. Some of the differences, according to Wright,[28] are:

> The metabolism of muscle maintaining posture is low compared to that of muscles executing movments: it is only 25% higher than that of completely paralyzed muscle. Postural muscle is also relatively infatiguable. Both of these features can be readily accounted for if the nature of the nervous discharge from the spinal cord is examined. If suitable electrodes are placed in small muscle fibre groups and the action currents recorded, it is found that the muscle is receiving impulses at a low rate, varying from 5-20%. We are therefore dealing with a slow discharge from the anterior horn cells. The muscle fibers thus stimulated pass into a state of partial tetanus.

The blood supply to the muscles is regulated partially by local products of activity and partially by a general adjustment of the circulation to the needs of the muscle. The blood supply of postural muscles is not so great as that of muscles which execute movements, because of the lower metabolism and activity of the postural muscles. In addition to these factors, it is known that the cervical muscles are subject to constant strain and are frequently held for a long time in the same position. Also, the neck is more exposed to changes in temperature than is any other portion of the body.

TREATMENT

In cases of subacute and chronic indurative headache it is necessary either to make a complete examination of the patient, keeping in mind the possibility of an endocrine disturbance and allergy, or to refer him to an internist for this service. Such general therapy as is found necessary by this examination should be instituted, not, however, with the expectation that this form of therapy in itself will cure or prevent attacks of indurative headache. Moreover, I have not found that the elimina-

27. Hunt, R. J.: Tr. A. Am. Physicians **51**:222, 1936.

28. Wright, S.: Applied Physiology, ed. 4, New York, Oxford University Press, 1932, p. 56; Proc. Roy. Soc. Med. **32**:651 (April) 1939.

tion of foods to which allergic patients are sensitive will prevent or cure this condition. The same may be said about the removal of foci of infection. I do believe, however, that proper attention to any or all of these factors will bring about a general improvement in the health of the patient and will reduce both the severity and the frequency of attacks. Antineuralgic agents and narcotics act merely as central depressants but are of value when severe pain is present.

Massage, in spite of its limitations, is still the best method of therapy available. Wright [28] stated:

> The movements employed in massage are differently described and classified by different authors. In a general way there are three main varieties of movements: stroking, compression, and perscussion movements. In the light of current physiological knowledge we may state that very light stimulation acts directly on the capillaries of the skin causing vasoconstriction. More vigorous stroking, especially in susceptible people, produces cutaneous vasodilation of varying degree. This cutaneous vasodilation is due to the direct action of histamine on the capillary wall. The arteriolar dilation is due to a local axon reflex; histamine stimulates sensory nerve endings and impulses pass through cutaneous nerve plexuses to reach adjacent arterioles. As a result, the blood flow through the skin is increased and the cutaneous temperature may rise from 1 to 2 degrees F. If the stroking is centripetal, the flow in the superficial veins and lymphatics may be hurried on, especially if the circulation is impaired. Nothing is known about the effects of massage on the rate of removal of fatigue or other noxious products. It is difficult to dogmatize about the reflex effects produced by massage. In experimental animals, sensory nerve stimulation produces effects which vary with the nerve used, the kind, strength, duration of stimulation and the state of the animal. The blood pressure may rise or fall, and the breathing may be profoundly altered. Similarly, in man, it may be supposed that the effects will depend on the exact mode of stimulation employed and will vary with different kinds of mechanical, thermal or chemical stimulation. General vigorous massage does not increase the O_2 consumption, CO_2 output, or heat of the muscles. There is no change in the blood lactate or inorganic phosphate. Chemical agents may be liberated or expressed into the blood and may produce local or general effects. In this connection it is worth drawing the attention again to the many metabolites, both known and unidentified, that are liberated during muscular activity or during muscle damage or ischaemia. Some or all of these might be liberated by massage.

Antiphlogistics tend to act directly on the process in the muscle. Unfortunately, however, as with massage, it is not always possible to predict what results will be obtained. The effect depends on a number of factors, all of which are little understood; e. g., the type of myalgia (acute or chronic), the degree of heat used, the duration of application and the type of nerves in the area treated. According to my experience, all forms of heat produce the same results. From personal experience and from observation of my patients, I have found that heat and massage will sometimes not only relax the spastic muscles which are being treated but, probably through the absorption of metabolites, bring about a feeling of well-being and relaxation. On another occasion, the same masseur,

apparently using the same technic on the same patient, may increase the cervical pain, in addition to making the patient feel ill at ease and irritable.

Aside from the fact that one can never be certain of the results that will follow from massage and heat, there are many other factors that make it highly desirable that some method of therapy other than these be found for the treatment of indurative headache. I shall mention only a few of these factors. Heat and massage are only measures of relief. In small towns or in the country it is impossible to obtain trained masseurs. In cases in which the condition is subacute or chronic it often requires many weeks to bring about relief. As there may be a recurrence at any time, the patient often becomes discouraged and seeks aid from some other physician or from a cultist. Patients will rarely take the time necessary to treat themselves and when they do so usually obtain only indifferent results, especially if the condition is chronic.

To those of my patients whom I cannot send to a masseur, I give the following instructions: 1. Heat the neck with an electric pad for five minutes. 2. Rub into the affected area as well as that surrounding it a small quantity of an ointment containing histamine (imadyl unction). 3. Again apply the heat for from twenty to forty minutes. 4. Dry the neck and apply talcum powder and massage. The amount of force used in massaging an individual patient will depend largely on the amount of pain evoked by its use. For those suffering acutely it is usually best not to institute massage at first but to limit the treatment to unction and heat. I instruct the patient to massage the nodular or spastic muscles in a centripetal direction. This treatment is followed by a cross massage, which may be carried out by grasping and rolling the muscle between the fingers or by massaging across the muscle with the fleshy part of the thumb. Next I have the patient exert considerable pressure on those areas in the muscle which are the most painful. This pressure is applied across the fibers of the muscle with the ball of the thumb. The pressure is exerted for only a few seconds at a time, with an interval of rest between the applications of pressure, and is repeated from three to ten times, again according to the sensitivity of the muscle. This maneuver may be repeated five or six times a day. Finally, I have him massage those cervical muscles that are not painful or in spasm. If the muscles of the neck are very sensitive on the day following the treatment it is better to wait another day before repeating the massage. These treatments must be carried out until all pain, nodules and spasms disappear. The patient should be informed that it is a fairly simple matter to obtain relief if he carries out the treatment first described soon after the pain in the head or neck appears. If, on the contrary, this treatment is postponed until the pain becomes severe, little immediate relief from the treatment may be expected. Under these conditions the patient must be put to bed and sedatives

administered in large doses. Frequently there will be a mild attack of pain, which if not relieved will increase in intensity each day until the suffering becomes almost unbearable.

For many years I have used, with success in certain cases, hypodermic injections of procaine hydrochloride. As a rule the best results are obtained in those cases in which the existing pain can be increased or duplicated by pressure on the so-called sensitive areas in the muscle. I have, however, obtained splendid results by injecting the drug into these areas even when the existing pain is not increased or reproduced. My technic for a number of years was to make injections into the skin covering these areas by using a fine gage hypodermic needle, subsequently infiltrating the muscles beneath the skin. Lately I have eliminated the deep infiltration, and although I have not as yet treated enough patients to warrant, certainty, I believe that my results are better than with the old technic.

For several years I have been anxious to determine the results that would follow blocking of the cervical sympathetic system in indurative headache. Since the patients I have seen have been in private office cases, I have not felt free to carry out this procedure. In addition to the methods of treatment already mentioned, I have experimented with the following drugs and methods of therapy: (*1*) acetylbetamethylcholine hydrochloride (for iontophoresis); (*2*) histamine given hypodermically in an attempt to immunize the patient; (*3*) ice used locally with the idea of desensitizing the patient; (*4*) ethyl chloride; (*5*) ergotamine tartrate; (*6*) ephedrine used both locally (injected into the nodules) and for its general effect; (*7*) histamine injected into the nodules; (*8*) roentgen therapy of the cervical portions of the sympathetic chain. My experience with these remedies has not resulted in any degree of success. I shall not give a detailed report, as I first wish to give these agents a more thorough trial.

SUMMARY AND CONCLUSIONS

A condition which is the cause of one of the most frequent forms of headache with which the otolaryngologist comes in contact is described.

This headache is often incorrectly diagnosed, being confused with that which accompanies sinusitis, otitis media, mastoiditis and migraine.

Indurative headache results from a myalgia of the cervical muscles.

The cause of myalgia has not yet been determined. The exciting factor seems always to be an exposure to cold and draughts.

The treatment consists of a definite type of massage and the injection of procaine hydrochloride into the painful areas in the neck.

It is hoped that this paper will stimulate others to work with the problem, so that in time the causative factors of myalgia and a method of therapy that will allow removal of these factors may be discovered.

A REVISION OF THE CEREBELLOPONTILE ANGLE LESION SYNDROME

WITH AN ANALYSIS OF THE VESTIBULAR FINDINGS IN THIRTY-FOUR CASES OF VERIFIED TUMOR OF THE CEREBELLOPONTILE ANGLE

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It is not the object of this paper to show that a vestibular examination is a valuable aid to the neurologist or neurosurgeon in the study of patients thought to have a lesion in the cerebellopontile angle. Apropos of this wrote Harvey Cushing[1] in his monograph entitled "Tumors of the Nervus Acusticus":

> It would be ideal could the lesion be recognized when the acusticus alone is involved; that is, before there is evidence of implications of the other structures in the recess; but so many disorders may cause tinnitus, labyrinthine vertigo and deafness that until the otologist comes to our aid and helps to differentiate the various conditions which occasion these symptoms, we are not likely to recognize in vivo the acoustic tumors at this early stage.

It is rather the object to determine, from an analysis of 34 cases of verified tumor of the cerebellopontile angle, if the condition is adequately expressed by the term "cerebellopontile angle lesion syndrome," as it appears in textbooks of otolaryngology and in the rest of the literature; that is, does the name apply typically to most cases of angle tumor, and if not, can the designation of the syndrome be changed so as to apply to a greater number of cases? Again, can it be foretold from the vestibular picture alone whether the lesion is a neural lesion, that is, one arising primarily from the eighth cranial nerve, or an extraneural lesion, that is, a lesion secondarily involving the eighth nerve?

Eugene R. Lewis[2] stated in Jackson and Coates' textbook, "The Nose, Throat and Ear and Their Diseases":

> A single lesion at or near the junction of the eighth nerve and brain stem frequently causes total abrogation of function of the cochlea, the horizontal and

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Read at the meeting of the Philadelphia Laryngological Society, Feb. 6, 1940.

1. Cushing, H.: Tumors of the Nervus Acusticus, Philadelphia, W. B. Saunders Company, 1918.

2. Lewis, E. R., in Jackson, C., and Coates, G. M.: The Nose, Throat and Ear and Their Diseases, Philadelphia, W. B. Saunders Company, 1929.

vertical canals of the same side, and of the vertical canals of the opposite side. This is called the cerebello-pontile angle lesion syndrome.

This is the accepted neuro-otologic syndrome for the diagnosis of a lesion of the cerebellopontile angle. Grant and Fisher [3] put essentially the same statement in other words: "In a cerebellopontile angle lesion, the only portions of the ear mechanism functioning properly are the cochlea and the horizontal canal of the good or unaffected side." Inasmuch as a loss of cochlear and vestibular function on the same side as the lesion is indicative of a peripheral lesion, and since a loss of function of the vertical semicircular canals with good function in the horizontal semicircular canal and the cochlea on the side opposite the lesion is indicative of a central lesion, they pointed out that in every case of lesion of the cerebellopontile angle two contradictory types of findings should be shown: (1) findings typical of a peripheral lesion and (2) those typical of a central lesion. When the vestibular picture in a given case conforms to that of the syndrome just described it is regarded as typical; however, when variations occur, the picture is regarded as atypical even when the two contradictory elements of a peripheral lesion and a central lesion are present. For example, on the opposite side there may be a loss of function of both the vertical and the horizontal semicircular canals with good cochlear function. This is also evidence of a central lesion, yet it renders the vestibular picture atypical of the cerebellopontile angle syndrome. Again, the contralateral eighth nerve (cochlear and vestibular) may be found to function normally, but the patient may have a spontaneous nystagmus on looking up, down or straight ahead or a horizontal nystagmus of central origin on looking extremely laterally; in addition, there may be a disproportionate reduction in the duration of nystagmus and vertigo after turning. These findings are also indicative of a central lesion. Variations may occur on the same side of the lesion. There may be no loss, little or moderate loss of hearing with no vestibular function. When there is little or no loss of hearing, the diagnosis cannot usually be made. Again, there may be complete deafness with some function remaining in the vestibular mechanism. Bilateral angle tumors never conform typically with the cerebellopontile angle lesion syndrome, in that there is evidence of a peripheral lesion on both sides. The central element of the picture must be supplied by a spontaneous nystagmus.

It has been the common belief that when the findings are typical of the cerebellopontile angle lesion syndrome, the anomaly is probably a lesion primarily arising from the eighth nerve in the angle, and conversely that when the findings are atypical of that syndrome, but never-

3. Grant, F. C., and Fisher, L.: J. f. Psychol. u. Neurol. **34**:113-185, 1926.

theless suggestive of a lesion in the angle, the anomaly is probably a lesion of the angle secondarily involving the eighth nerve. Thus, Grant and Fisher [8] in writing of their analysis of 30 cases of verified cerebellopontile angle lesion, made the following statement:

> It is interesting to note from an analysis of these reports that the typical phenomenon complex is, as was expected, more often encountered in true acoustic nerve tumors, while in extra-neural angle tumors both horizontal and vertical canals of the opposite functioning ear tend to show impaired response.

If these premises are true, there could be no justification in changing the designation "cerebellopontile angle lesion syndrome."

My series of 34 cases were all studied at the University Hospital, in the services of Dr. Charles H. Frazier, Dr. William G. Spiller and Dr. Francis C. Grant, between 1927 and 1939.

One patient, Mrs. M. P., was seen in the ophthalmic service of Dr. Thomas B. Holloway before being transferred to the service of Dr. Charles H. Frazier. Mrs. W. D. G. was seen outside of the hospital for vestibular examination before being admitted to the service of Dr. Francis C. Grant. In 1 case, that of Mr. L. R., who refused operation at the University Hospital, the diagnosis was verified at the Johns Hopkins Hospital, Baltimore, by Dr. Walter E. Dandy. In another case, that of Mrs. C. B. M., the diagnosis was verified by Dr. Temple Fay, at Temple University Hospital. A pathologic report was obtained in all cases except in that of 1 patient, Mrs. C. B. M., in whom a cyst in the angle was found; such a lesion is usually regarded as a glioma. The pathologic report served as a basis for classifying the cases into (1) a group in which the lesions were neural and (2) one in which they were extraneural. Each of these two groups was subdivided into (*a*) a group in which the findings were typical of the cerebellopontile angle lesion syndrome and (*b*) one in which they were atypical. In the group with typical findings were included those cases in which there was slight or complete loss of function of the opposite vertical semicircular canals, with good function but obliquely perverted response in the horizontal semicircular canal. Four cases in which the hearing was almost but not completely gone on the side of the lesion were also included (table 1, F. G. McM.; table 2, T. G., C. C. and S. M. H.). In the group of cases with findings atypical of the "cerebellopontile angle lesion syndrome" were included those cases in which the opposite vertical and horizontal semicircular canals were nonfunctioning and those in which the opposite vertical and horizontal semicircular canals functioned normally. Four cases in which there was some residual function in the homolateral vestibular apparatus were also included in this group (table 1, M. O. and J. B. D.; table 2, M. C. and C. B. M.).

TABLE 1.—*Cases of Neural Lesions*

Case No.	Patient	Homolateral Vestibular Function: Vertical Canals	Homolateral Vestibular Function: Horizontal Canal	Hearing	Vestibular Contralateral Function: Vertical Canals	Vestibular Contralateral Function: Horizontal Canal	Romberg's Sign	Pelvic Girdle Movements
1	H. S.	None	None	Deaf	None	Oblique nystagmus up and to L	Negative	.....
2	W. A. K.	None	None	Deaf	None	Oblique nystagmus up and to L	Positive	.....
3	P. S.	None	None	Deaf	None	Oblique nystagmus up and to L	Positive	Poor
4	O. S.	None	None	Deaf	None	Oblique nystagmus up and to R	Positive	Poor
5	E. S.	None	None	Deaf	None	Oblique nystagmus up and to R	Positive	Poor
6	F. N.	None	None	Deaf	None	Oblique nystagmus up and to R	Positive	Poor
7	L. R.	None	None	Deaf	Rotatory nystagmus to R, fair 3 min.	Oblique nystagmus up and to R	Positive	Poor
8	R. F.	None	None	Deaf	Rotatory nystagmus to R and oblique down and to R, good 85 sec.	Oblique nystagmus up and to R	.....	.....
9	S. B.	None	None	Deaf	Good 105 sec.	Oblique nystagmus up and to R	Positive	Poor
10	F. G. McM.	None	None	Markedly impaired	Rotatory nystagmus to L, good 90 sec.	Oblique nystagmus up and to L	Positive	Poor
11	L. R.	None	None	Deaf	Rotatory nystagmus to L, good 75 sec.	Oblique nystagmus up and to L	Negative	Impaired to left
12	W. H. C.	None	None	Markedly impaired	Rotatory nystagmus to L, fairly good 30 sec.	Oblique nystagmus up and to L	Positive	Poor
13	W. D. G.	None	None	Markedly impaired	Rotatory nystagmus to R, good 35 sec.	Oblique nystagmus up and to R	Positive	Poor to right
14	W. M.	None	None	Deaf	Rotatory nystagmus to R, good 40 sec.	Oblique nystagmus up and to R	Positive	Poor
15	M. O.	None	Oblique nystagmus up and to L and horizontal to L, poor	Deaf	None	Oblique nystagmus up and to R	Positive	.....
16	M. P.	None	None	Deaf	Rotatory nystagmus to L, good 40 sec.	Oblique nystagmus up and to L	Positive	.....
17	R. D.	None	None	Deaf	Rotatory nystagmus to L, good 25 sec.	Horizontal nystagmus to L	Negative	Good
18	J. B. D.	Rotatory nystagmus to R, good 60 sec.	Horizontal nystagmus to R, fair	Markedly impaired	Rotatory nystagmus to L, good 2¼ min.	Horizontal nystagmus to L	.....	.....
19	B. W.	None	None	Deaf	Rotatory nystagmus to R, fairly good 50 sec.	Horizontal nystagmus to R	Negative	Good

Spontaneous Nystagmus					Duration of Nystagmus After Turning in Both Directions	Duration of Vertigo After Turning in Both Directions	Vestibular Picture
Looking Up	Looking Down	Looking Right	Looking Left	Looking Ahead			
None	Rotatory to R; horizontal to R	None	None	Vertical up; horizontal to R	.....	.....	Typical
Vertical up	None	Horizontal to R	Horizontal to L	None	Different	Different	Typical
Vertical up	None	Horizontal to R	Horizontal to L	None	Different	Same	Typical
Vertical up O. D.	None	Horizontal to R	Horizontal to L	Horizontal to L	Different	Same	Typical
Vertical up	None	Horizontal to R	None	None	Different	Different	Typical
Vertical up	None	Horizontal to R	Horizontal to L	None	Same	Same	Typical
Vertical up	None	Horizontal to R	Horizontal to L	None	Different	Different	Typical
Vertical up	None	Horizontal to R	Horizontal to L	None	.....	.....	Typical
Horizontal to L	None	Horizontal to R	Horizontal to L	Horizontal to L	Different	Different	Typical
Rotatory to R; vertical up	Rotatory to L	Horizontal to R	Horizontal to L	None	Different	Different	Typical
None	Rotatory to L	Horizontal to R	Horizontal to L	None	Same	Same	Typical
None	Rotatory to L	None	Horizontal to L	None	Different	Different	Atypical
Oblique up and to L; rotatory to L	Horizontal to L; oblique down and to L	Horizontal to R	Horizontal to L	None	Different	Different	Atypical
Vertical up	None	Horizontal to R	Horizontal to L	None	Different	Different	Atypical
Vertical up	None	Horizontal to R	Oblique up and to L	None	Different	Different	Atypical
Vertical up	None	Horizontal to R	Horizontal to L	None	Same	Different	Atypical
Rotatory to R	Rotatory to R; horizontal to R	Horizontal to R	Horizontal to L	None	.....	.....	Atypical
None	None	Horizontal to R	Horizontal to L	None	Different	Same	Atypical
Vertical up	Rotatory to R	Horizontal to R	Horizontal to L	Horizontal to L	Different	Different	Atypical

The cases of bilateral angle tumor were listed separately (table 3) and were divided into a group in which the lesions were neural and one in which they were extraneural. As has previously been stated, bilateral angle tumors do not conform with the cerebellopontile angle lesion syndrome. One case (table 3, Mr. J. W.), in which a small neuroma on the left side was removed and no tumor found on the right side, was included in the neural group of cases of bilateral angle tumor. The patient in this case died as a result of a brain tumor in the Allentown

TABLE 2.—*Cases*

Case No.	Patient	Homolateral Vestibular Function: Vertical Canals	Homolateral Vestibular Function: Horizontal Canal	Hearing	Vestibular Contralateral Function: Vertical Canals	Vestibular Contralateral Function: Horizontal Canal	Romberg's Sign	Pelvic Girdle Movement
1	T. G.	None	None	Markedly impaired	None	Oblique nystagmus up and to L	Negative	Good
2	J. K.	None	None	Deaf	None	Oblique nystagmus up and to L	Positive	.....
3	C. C.	None	None	Markedly impaired	None	Oblique nystagmus up and to L	.....	.....
4	M. S.	None	None	Deaf	None	Oblique nystagmus up and to L	Positive	.....
5	S. M. H.	None	None	Markedly impaired	Rotatory nystagmus to R, fairly good 90 sec.	Oblique nystagmus up and to R	Negative	Poor
6	M. C.	None	Horizontal nystagmus to L, fair	Deaf	None	Oblique nystagmus up and to R	Positive	.....
7	C. B. M.	None	Nystagmus, rotatory to R; oblique up and to R, fair	Impaired	Rotatory nystagmus to L, good 3 min.	Nystagmus, vertical up; oblique up and to L	Positive	.....
8	K. M.	None	None	Deaf	Rotatory nystagmus to L, good 35 sec.	Oblique nystagmus up and to L	Positive	.....

State Hospital nine months after operation. The vestibular examination revealed a totally nonfunctioning eighth nerve (cochlear and vestibular) on both sides, with a spontaneous nystagmus when the patient looked in any direction. In another case (table 3, Mrs. B. D.), a single large meningioma was found at autopsy. The tumor arose from the falx cerebri and extended over the optic chiasm, causing an atrophy of this structure. It then extended posteriorly to the pons, compressing and flattening it out. The patient in this case showed a loss of function of the eighth nerve (cochlear and vestibular) on both sides and on looking straight ahead showed a spontaneous mixed rotatory and horizontal nystagmus to the right.

Of the 34 cases, 27 were of unilateral tumors and 7 of bilateral. Of the cases of unilateral tumors, 19 were of neural and 8 of extraneural lesions. Of the 7 cases of bilateral angle tumor, 5 were of neural and 2 of extraneural lesions.

Of the 19 cases of unilateral neural lesions, in 11, or 57.8 per cent, the findings were typical, and in 8, or 42.2 per cent, atypical, of the cerebellopontile angle lesion syndrome.

traneural Lesions

Spontaneous Nystagmus					Duration of Nystagmus After Turning in Both Directions	Duration of Vertigo After Turning in Both Directions	Vestibular Picture	Type of Lesion
Looking Up	Looking Down	Looking Right	Looking Left	Looking Ahead				
None	None	Horizontal to R	None	None	.....	.....	Typical	Chondroma from cervical portion of spine
Vertical up	None	Horizontal to R	Horizontal to L	None	Same	Different	Typical	Astrocytoma of brain stem
Vertical up	None	Horizontal to R	None	Rotatory to R; horizontal to R	.....	.....	Typical	Glioma of pons
Vertical up	Horizontal to L	Horizontal to R	Horizontal to L	Horizontal to L	Different	Different	Typical	Astrocytoma of brain stem
Vertical up; oblique up and to L	None	Horizontal to R	Horizontal to L	None	Different	Different	Typical	Endotheli-oma
Vertical up	None	Horizontal to R	Horizontal to L	None	Different	Same	Atypical	Metastatic carcinoma
Vertical up	None	Horizontal to R	Horizontal to L	O.D., rotatory to R; O.S., vertical up	Different	Different	Atypical	Cyst, left angle
Oblique up and to R	Vertical down	Horizontal to R	Horizontal to L	None	Different	Same	Atypical	Primary cholesteoma

Of the 8 cases of extraneural unilateral angle tumor, in 5, or 62.5 per cent, the findings were typical and in 3, or 37.5 per cent, atypical.

When the neural and the extraneural unilateral angle tumor group were combined, the typical picture was seen in 16 cases, or 59.2 per cent. Thus, the typical picture was found somewhat more frequently than the atypical in both the neural and the extraneural group. The typical picture was seen somewhat less frequently in the neural than in the extraneural group.

In Grant and Fisher's series of 30 cases of verified lesions in the cerebellopontile angle, there were 13 cases of neural and 17 of extraneural angle tumors. In the neural group, the typical picture was seen

TABLE 3.—*Five Cases of Bilateral Angle Tumors*

Case No.	Patient	Vestibular Function: Right: Vertical Canals	Vestibular Function: Right: Horizontal Canal	Vestibular Function: Left: Vertical Canals	Vestibular Function: Left: Horizontal Canal	Hearing	Spontaneous Nystagmus: Looking Up	Looking Down	Looking Right	Looking Left	Looking Ahead	Romberg's Sign	Vestibular Picture	Type of Lesion
							Neuromas							
1	G. F.	None	None	None	None	Deaf bilaterally	Vertical up	Vertical down	Horizontal to R	Horizontal to L	None	.....	Atypical	Neuroma
2	B. W.	None	None	None	None	Deaf bilaterally (blind)	?	?	?	?	None	Positive	Atypical	Neuroma
3	A. K.	Practically none	None	Practically none	None	Deaf bilaterally	Vertical up	None	Horizontal to R	Horizontal to L	None	Positive	Atypical	Neuroma
4	A. Z.	None	None	None	None	Deaf bilaterally	Vertical up	Rotatory to R	Horizontal to R	Horizontal to L	None	Positive	Atypical	Neuroma
5	J. W.	None	None	None	None	Deaf bilaterally	Vertical up	Vertical down	Horizontal to R	Horizontal to L	None	Positive	Atypical	Neuroma
							Extraneural Lesions							
1	M. W.	None	None	None	None	Deaf bilaterally (blind)	None	None	Horizontal to R	Horizontal to L	None	.....	Atypical	Medulloblastoma, both cerebellar hemispheres, involving angles
2	B. D.	None	None	None	None	Deaf bilaterally (blind)	?	?	?	?	Rotatory to R; horizontal to R	.....	Atypical	Meningioma from falx cerebri, extending to and compressing pons

in 10 cases, or 76 per cent, and in the extraneural group in 11, or 64 per cent. In their combined neural and extraneural group of 30 cases, the typical picture was seen in 21, or 70 per cent.

In practically all of my cases there were (1) a spontaneous nystagmus on the patient's looking up, down or straight ahead; (2) a disproportion between the duration of nystagmus and vertigo after turning, and (3) a loss of function of either the vertical semicircular canals or the vertical and horizontal semicircular canals with good hearing on the opposite side. These findings are indicative of a central lesion. In all of my cases there was tolerance to vestibular stimulation. In 7 of my cases of unilateral angle tumor the opposite vertical and horizontal semicircular canals functioned normally, but in these cases there was a spontaneous nystagmus which supplied the central element of the vestibular picture.

If the term cerebellopontile angle lesion syndrome should be changed to "evidence of a peripheral lesion on the same side as the cerebellopontile angle lesion in addition to evidence of a central lesion" (the latter evidence not being confined to a loss of function of the opposite vertical semicircular canals), then in 23, or 85.1 per cent, of my 27 cases of unilateral angle tumor the condition would be considered as conforming typically with the revised concept of the syndrome associated with a lesion of the cerebellopontile angle. In 6 of the 7 cases of bilateral angle tumor also, the condition would conform typically to the revised concept. In 1 case of bilateral angle tumor (table 3, B. W.), it was not possible to make an examination for spontaneous nystagmus, for the patient was bilaterally totally deaf and blind. In both cases of extraneural bilateral angle tumor, bilateral total deafness and blindness were also present, but spontaneous nystagmus could be observed in 1 case, when the patient looked straight ahead and in the second case when the patient deviated the eyes to one side or the other. Combining the group of unilateral and that of bilateral angle tumors, one might say that the typical picture was found in 29 of the 34 cases, or 85.3 per cent.

SUMMARY AND CONCLUSIONS

It is shown that according to this analysis of 34 cases of verified cerebellopontile angle tumor, the typical syndrome, as it is generally understood, appears somewhat more frequently in both neural and extraneural angle tumor groups. It appears somewhat more frequently in the extraneural group than in the neural. One is not justified, therefore, in foretelling from a vestibular examination alone whether the anomaly is a primary or a secondary lesion of the angle.

It is also pointed out that the condition designated as "cerebellopontile angle lesion syndrome" was typically demonstrated in 59.2 per

cent of the cases of unilateral angle tumor. The term does not apply in a typical manner when a bilateral angle lesion is present. If the designation of the syndrome were changed to "evidence of a peripheral lesion on the same side as the cerebellopontile angle lesion in addition to evidence of a central lesion," then 85.3 per cent of the 34 cases of this series could be said to conform in a typical manner to the revised concept of the syndrome associated with a lesion of the cerebellopontile angle.

1930 Chestnut Street.

RADIATION THERAPY FOR CARCINOMA OF THE LARYNX

OBSERVATIONS AFTER TWENTY YEARS

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There are so many aspects to the story of radium as a therapeutic agent in cases of carcinoma of the larynx that I find it difficult to array the items in their logical sequence. And yet it was my purpose in preparing this paper to set forth the evidence as I have found it in twenty years' observation, hoping thereby to crystallize such conclusions as mature deliberation might evolve. It is not an easy task, because the mystery and glamour which surrounded this element from the time of its discovery still seem to captivate the imagination of both physicians and the public to a degree that is incompatible with the exercise of cool judgment. The hope which it inspired in the human breast, that at last the dreadful scourge of cancer was to be lifted through this powerful agent, was too thrilling to be easily abandoned or even modified by sober experience. Much of this feeling, I believe, may be attributed to the attitude of the press, which always seeks to capitalize the sensational. Startling headlines and incomplete quotations of responsible physicians in connection with the hopes and promises of radiation therapy have done much to foster an optimism which is out of proportion to accomplished facts. The mere account of the establishment of a new cancer clinic, stressing the high cost of the radium or the magnitude of the high voltage machines, must necessarily impress the average layman and lead to the conclusion that such a valuable armamentarium possesses therapeutic merit commensurate with its cost. This is a psychologic phenomenon which is difficult to counteract, since it is natural for the untrained mind to judge values in direct proportion to costs. When with this faith in the potency of a mysterious costly agent (which has in some quarters been exploited) one considers the average resistance to major surgical procedures, especially when they concern a conspicuous organ like the larynx, it is not difficult to appreciate the advantage which this nonsurgical therapy possesses in the layman's mind. And that brings me to the first point which I hope to demonstrate before I have finished,

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namely, that only under certain conditions is there such a thing as a choice between operation and irradiation. Many patients honestly believe that such a choice is possible regardless of the status of the lesion.

Lest I be accused of prejudice in this matter, let me say that I have the highest regard for the professional integrity and scientific attainments of the many hard-working, sincere investigators in the field of irradiation and have profited much by their advice and cooperation. Theirs has not been an easy task—having to struggle with an agent whose potentialities were not fully understood, with a public that demanded impossible accomplishments and with the odium cast unjustly on them by a few unscrupulous and half-baked therapeutists.

The early days of radium therapy are like a bad dream. In the frantic hope of saving a patient's life or sparing him an operation, heavy radium needles were thrust into the tumor mass and its bed, then bare glass emanation seeds, gold seeds and platinum seeds. Or intubation tubes and two way tracheal tubes were used as carriers for the feeble and futile milligrams of radium. In addition, the patients were burned from without with inadequate, ineffective and improperly screened element or emanation. When I worked with Freer in the early twenties I was fired with the contagion of his enthusiasm for what we thought was a solution to the problem, namely, a very elaborate emanation carrier by which as much as 500 millicuries could be introduced within the larynx without trauma. But all in vain! The incidence of burns and necrosis was so high that very few observers had the courage to tell the whole truth at that time. In their recent book, Jackson and Jackson,[1] comparing results prior to 1930 with those in a subsequent period, listed a series of 106 cases of irradiation in the former period, in 32 of which serious complications, such as sloughing and necrosis, developed. I am sure that all physicians are familiar with this picture. As for the actual percentage of cures, when reliable statistics were available there was little cause for rejoicing. Lewis,[2] in 1921, reported to this society a collection of 109 published cases, in which only 10 patients survived for variable periods. Greene,[3] in 1920, reported 30 cases in which the patients were treated by various technics and in which the longest survival was for two years. Imperatori,[4] in 1926, reported 30

1. Jackson, C., and Jackson, C. L.: Cancer of the Larynx, Philadelphia, W. B. Saunders Company, 1939.

2. Lewis, F. O.: Radium in the Treatment of Carcinoma of the Larynx with a Review of the Literature, Ann. Otol., Rhin. & Laryng. **30**:942 (Dec.) 1921.

3. Greene, D. C.: The Results of Treatment of Malignant Disease of the Upper Air Tract by Radiation, Tr. Am. Laryng. A. **42**:73, 1920.

4. Imperatori, C. J.: An Analysis of Thirty Cases of Carcinoma of the Larynx and Laryngo-Fissure Treated with Radium, Arch. Otolaryng. **4**:151 (Aug.) 1926.

cases, in 1 of which the patient was cured because the entire lesion was so small that it was probably extirpated at the time the biopsy specimen was taken. He also reported the survival of another patient on whom a laryngectomy was performed after unsuccessful needling. In 1925, I [5] reported the results in 40 cases of cancer treated by Freer and myself, in which only 3 patients survived without recurrence for as long as three years. Quick and Johnson's [6] results, published in 1923, were better than those just mentioned for intrinsic cancer, although the periods over which they were observed were hardly long enough to warrant the assumption that the lesions were cured. As for the continental authors, I do not care to cite their statistics, because in many instances their conclusions have been based on inaccurate premises. In fact, to this day I find that some of them figure their percentage of cures on the basis of the number of patients that were followed up, eliminating those that could not be traced. This is a species of self delusion that I cannot quite fathom.

In the early days of irradiation therapy comparatively little thought was given to the question of proper indications. Cancer clinics were the dumping grounds for patients with far advanced lesions who came in from all points of the compass, in many instances because the attending physicians wished to get them off their hands, in other cases impelled by the vain hope that the new remedy might be their salvation and in still others frankly in response to misdirected and overenthusiastic publicity. In many cases no biopsy was done. Naturally even the best therapeutists had to struggle with this problem, experimenting with various technics in the face of repeated disaster and hoping that some day they might be given the opportunity of competing with the surgeon on an equal basis by having access to the early intrinsic lesions. There were others, less scrupulous, who did not hesitate to issue exaggerated statements which might easily be considered misleading. I quote the following comments from a book on radium therapy published within the last twenty years: "Radium is the agent of choice for the treatment of carcinoma of the larynx because it can be applied directly against or to the growth." "Laryngectomy is mutilating. However, with excellent surgical technique the curative results are superior to those with radiation. The same result can be accomplished without mutilating operations and with a minimum of danger to the patient." A more inconsistent and provocative statement was never written. With the advent of the protracted fractional irradiation technic of Regaud and Coutard, which

5. Salinger, S.: Radium in Carcinoma of the Larynx, Arch. Otolaryng. **1**:286 (March) 1925.

6. Quick, D. A., and Johnson, F. M.: Treatment of Malignant Neoplasms of the Larynx, Am. J. Roentgenol. **9**:599 (Sept.) 1932.

permitted delivery of hitherto unheard-of doses at a reduced risk, radiologists and radium therapeutists acquired increased confidence in their mediums and soon began treating these early lesions. Some conclusions may perhaps be drawn from the cases to be presented here.

Having mentioned the question of indications for therapy, it will be advisable to examine the question more closely. Regaud was one of the first to stress the radiosensitivity and radioresistance of different types of cells. Since a great many tumors of the throat consist of anaplastic cells, which were proved to be particularly sensitive to gamma rays, it was taught that where these cells predominated the prognosis through irradiation would be good. Consequently, for a number of years a great deal was written on this phase of the question, and the indications for therapy were based principally on histologic observations. Gradually it became apparent, however, that in dealing with lesions about the larynx other factors had to be taken into consideration, particularly the seat of the lesion, that is, the tumor bed and the intervening cartilage. And so the histologic structure of the growth, instead of being the sole therapeutic indication, had to be taken in connection with the other factors, which consequently led to new dictums. Coutard [7] has stated that he now draws the line at the vocal cords. He has claimed that in this area and below it the cells are more highly differentiated and have a tendency to grow into the muscle tissue, causing fixation, which, he stated, is a definite contraindication to irradiation. This is in contradistinction to tumors above the level of the true cords, which are more likely to be anaplastic and which develop in a stroma that is looser and more yielding. Yet the matter is not as simple as it sounds. Coutard,[8] in analyzing his results year by year from 1922 to 1938, was at a loss to explain why he had good years and bad years irregularly interspaced. This perplexity was the reason for his ever changing technic, varying the amount of radiation delivered per day, the size and location of the portals and the number of days of exposure, so that one could never say definitely that this or that was the proper technic. Even at present there is a total lack of unanimity in various centers on the subject of dosage except in the general agreement that the maximum compatible with the tolerance of the tissues must be given. This permits considerable latitude for the exercise of individual judgment in individual cases. Coutard admitted that his vacillation between a high total dose over a long period and a low total dose over a shorter period but with

7. Coutard, H.: Results Obtained by Ray Therapy, Tr. Am. Laryng., Rhin. & Otol. Soc. **44**:162, 1938.

8. Coutard, H.: Cancer of the Larynx, Am. J. Roentgenol. **40**:509 (Oct.) 1938.

a higher daily dose has been due to a lack of understanding of how to apply them, since the two methods sometimes succeeded and sometimes failed.

Hautant's [9] excellent treatise, published in 1936, while stressing the question of radiosensitivity, also wisely gave due weight to consideration of the location and extent of the lesion. The author pointed to the value of roentgenographic study in determining the extent of the growth and said that, though a tumor which involves the vestibule and ventricular bands, even if bilateral, may generally present favorable indications for laryngectomy, there are some instances in which roentgenograms reveal extension to the preepiglottic space. This would indicate involvement of the anterior lymphatic nodes which would lessen the chances for a cure by this procedure. Also, roentgenograms might reveal involvement of the laryngeal cartilages, which would be a contraindication to irradiation regardless of the type of cell. A lesion limited to the vocal cord, said Hautant, may be amenable to irradiation, but operation is preferable because it is simpler, healing is more rapid and one still has irradiation as a reserve measure in case of recurrence. Subglottic tumors, being generally of differentiated cellular type and frequently invading the cartilage, are also unfavorable for irradiation.

On the other hand, Harris and Klemperer,[10] analyzing 32 cases of carcinoma of the larynx treated according to the principles of Coutard to determine the value of histologic structure as a basis of differentiation between radiosensitive and radioresistant tumors, came to the conclusion that the histologic structure is of minor importance. After considering the grade of cell, the mitotic count, cellular anaplasia, stroma reaction and location of the neoplasms, they were unable to establish any clearcut criteria as a basis for the prognosis of irradiation. Furthermore, they cited several foreign observers who have reached the same conclusions. It would seem, therefore, that at present the question of radiosensitivity is of less importance than the other factors mentioned and comes into consideration chiefly with lesions involving the introitus laryngis; that is, the lesions in those borderline cases in which the prospects of cure by laryngectomy are not good because of the anaplastic nature of the growth and the danger that the lymphatics may already have been invaded.

Before taking up in detail an analysis of results of irradiation therapy, let me point out that the danger of radiation necrosis has not been

9. Hautant, A.: La radiothérapie des cancers intra-laryngés. Ses indications et celles de la chirurgie, Ztschr. f. Hals-, Nasen- u. Ohrenh. **40**:15, 1936.

10. Harris, W., and Klemperer, P.: Pathologic Differentiation Between Radio-Sensitive and Non-Radio-Sensitive Malignant Neoplasms of the Larynx, Tr. Am. Laryng., Rhin. & Otol. Soc. **44**:148, 1938.

entirely eliminated despite increased knowledge of the medium and increased skill in its application. It must never be forgotten that this danger still threatens every patient whose larynx is exposed to heavy

Persistent submental brawny edema after subsidence of perichondritis. The soft tissues over the larynx are dense and adherent.

doses of radium or roentgen rays and that the disaster it entails constitutes a picture which, once seen, is never forgotten.

Hautant [9] studied the subject of radionecrosis very thoroughly and assigned four different explanations for its occurrence. His first explanation is that it is due to therapy badly distributed or given in too

large doses. He found the site of predilection to be the inferior portion of the anterior angle of the thyroid cartilage, a region where ossification is most advanced. In the passage of the rays the bone, composed of a large number of molecules of calcium, absorbs the element and gives rise to secondary radiations, which are unfiltered and therefore harmful. The bone is devitalized and becomes the seat of secondary infection leading to necrosis. He was so impressed with the magnitude of this complication that for a long time he practiced preliminary resection of this portion of the thyroid shield. The other explanations offered by Hautant for this complication are (1) that it is the result of radiation in cartilage already invaded by the carcinoma; (2) that recurrence of the growth, particularly in the anterior commissure, sets up a portal of entrance for secondary infection into the cartilage already weakened by the previous irradiation, and (3) that it may result from an acute laryngitis occurring during the postradiation period, while the cartilage is still recovering from the bombardment.

I have observed a number of cases of radiation necrosis in the past twenty years and can describe the process as one that inflicts the utmost in suffering that is humanly conceivable. Even in the past year I have had 2 patients with this awful complication, 1 of whom mercifully passed away within a few months; the other is still carrying on, God knows how. I should like to go more into detail because I believe there is much food for thought in the consideration of unfavorable results.

REPORT OF CASES

A 60 year old man complaining of hoarseness off and on for the past five years presented bilateral hypertrophy of both vocal cords and ventricular areas which restricted the glottic space and only partially interfered with the mobility of the cords. There were no palpable glands and no local tenderness, and the patient's general condition was excellent; so excellent, in fact, that when the biopsy revealed a squamous cell carcinoma the conditions seemed ideal for laryngectomy. The patient, however, stubbornly refused to consent, even when told bluntly that his life expectancy without operation could not exceed two years. He was willing to have a tracheotomy and irradiation therapy, despite the fact that little encouragement was given for a favorable outcome. A full course of radium bomb therapy was administered, the patient receiving 207,000 milligram hours through two fields (17,000 r) over a period of forty-four days. The treatment was completed April 4, 1939. There were the usual epithelitis and epidermitis, which subsided normally, and the lesion for a time seemed to give evidence of regressing. However, on June 19, a little over two months after completion of the treatment, edema and a brawny swelling appeared over the thyroid and submental area, accompanied by pain. At the same time the epiglottis and the folds became edematous. Within a few weeks the condition became progressively worse; there were foul breath, expectoration of blood and increasing pain. In an effort to relieve the pain, external incisions were made over the lateral thyroid regions, exposing foul, necrotic tissue overlying the cartilage. For a month or two, masses of necrotic slough were expelled through

these incisions, which alternately were open and closed. Direct laryngoscopic examination in September revealed massive edema of the larynx, obliterating all landmarks, and a slough, which was removed. Since the pain was still intense, the external incision was enlarged, and masses of slough and granulation tissue were removed, which on section proved to be malignant. The pain was relieved to a definite degree, and the external wound contracted. At the time of this report, however, the fistula is still open; the submental edema is still dense and firm, and the laryngeal introitus is completely closed by massive edema. Swallowing is still possible for fluids, but the patient is slowly losing ground, and the end is inevitable. Analyzing this case from the radiotherapeutist's point of view, one finds two favorable items, namely, a stricIty intrinsic lesion with mobility still present as against the one unfavorable factor, namely, a grade 1 squamous cell tumor. So far as the technic was concerned it was exactly the same as has been employed in numerous similar cases and was tolerated with no more distress than the average. On the basis of past experience the least one could expect was a palliative result. Instead, a complication occurred which it had been thought could not occur under present methods of treatment.

The second patient was a 45 year old man complaining of hoarseness for several months. He had swelling and fixation of the right arytenoid cartilage and swelling of the right ventricular band and cord with apparently some filling of the piriform fossa. It seemed as though the lesion, originating in the ventricular area, was either infiltrating through to the fossa or causing secondary edema of this area. Also the posterior surface of the epiglottis was ulcerated in its lower half, the upper half being only secondarily reddened and slightly thickened. There were no palpable glands, and biopsy revealed a squamous cell carcinoma with some transitional features. This, I believe, would be considered a borderline lesion. The possible involvement of the piriform fossa might predispose to recurrence after laryngectomy, yet the adult type of tumor cell would favor a good result from adequate surgical treatment. On the other hand, irradiation had little to offer, because experience has shown that squamous lesions of the epiglottis, infiltrative lesions reaching the piriform fossa, seldom yield to this form of therapy. I felt that the patient had a better chance with a laryngectomy and so advised him. The patient absolutely refused, despite the poor prognosis offered by irradiation. He was given a full course of radium bomb treatments by the protracted fractional method, receiving a total of 197,000 milligram hours (16,200 r) over a period of fifty-fours days, through two portals. There was practically no regression, and within two months after treatment he gave the usual evidence of having chondro-osteitis of the laryngeal box, massive edema, excruciating pain and dysphagia, leading to a very rapid decline and resulting in death five months after completion of the treatment.

My conclusions after pondering over these 2 cases are (1) that protracted fractional irradiation is still far from a perfected technic; (2) that infiltrating squamous cell lesions are poor risks for irradation, at least in curative doses, and (3) that my patients are the most stubborn in the world! Since the risk of chondronecrosis is not entirely eliminated, I am wondering whether in cases such as I have described it is worth while to subject the patient to heavy doses of irradiation and whether it would not be better to permit only such amounts as are considered palliative. No doubt there will be a difference of opinion on this point.

Certainly my experience is not singular, since the literature is full of references to this dangerous complication. The following authors may be cited: Iglauer [11] (1927), Clerf [12] (1927), Arbuckle [13] (1934), Halberstädter [14] (1932), Von Eicken [15] (1932), Uffenorde [16] (1932), V. A. Hutten [17] (1932), Seiffert [18] (1932), Hautant [9] (1936), Nelson and Hirsch [19] (1935), Martin [20] (1935) and Torrigiani and Palumbo [21] (1936 and 1939). Since the risk of this serious complication still exists, the question arises whether the results as a whole justify assuming this grave responsibility. Examination of the records is indicated. In our clinic there were 27 instrinsic lesions without palpable glands treated with radium, 6 of which were apparantly cured; 3 of the recovered patients I have been unable to trace beyond one year and the others have passed the five year mark. Of 4 patients with intrinsic lesions and palpable glands, 1 has remained well now over four years. Forty-seven extrinsic lesions were treated; 4 of the patients survived the five year period free from the disease. We had 9 patients who received postoperative irradiation either for recurrences or incomplete surgical treatment, 3 of whom survived with complete regression but are still under the five year period.

Schintz and Zuppinger [22] reported on 33 patients with intrinsic lesions, 6 of whom were free from symptoms for three years and 4 for five years. Their results with extrinsic lesions were as follows: There were 67

11. Iglauer, S.: X-Ray Injury to the Larynx with Report of a Case, Ann. Otol., Rhin. & Laryng. **36**:124 (March) 1927.

12. Clerf, L. H.: Laryngeal Complications from Irradiation, Tr. Am. Laryng. A. **49**:99, 1927.

13. Arbuckle, M. F.; Cowdry, E. V., and Votaw, R.: The Effect of Radium Emanations on the Laryngeal Cartilage, Tr. Am. Laryng. A. **56**:214, 1934.

14. Halberstädter, in discussion on papers by Holthusen, Hegener and Englmann, Mittermaier, and others, Ztschr. f. Hals-, Nasen- u. Ohrenh. **31**:263, 1932.

15. von Eicken, in discussion on papers by Beck and Klestadt and Fürst, Ztschr. f. Hals-, Nasen- u. Ohrenh. **31**:290, 1932.

16. Uffenorde, in discussion on papers by Beck and Klestadt and Fürst, Ztschr. f. Hals-, Nasen- u. Ohrenh. **31**:291, 1932.

17. Hutten, V. A., in discussion on papers by Beck and Klestadt and Fürst, Ztschr. f. Hals-, Nasen- u. Ohrenh. **31**:292, 1932.

18. Seiffert, in discussion on papers by Holthusen, Hegener and Englmann: Strahlentherapie in der Laryngologie, Ztschr. f. Hals-, Nasen- u. Ohrenh. **31**:129, 1932.

19. Nelson, P. A., and Hirsch, E. F.: Roentgen Radiation Necrosis of Larynx and Other Structures of the Neck, J. A. M. A. **104**:1576 (May 4) 1935.

20. Martin, H. E.: The Fractional or Divided Dose Method of External Irradiation in the Treatment of the Pharynx, Tonsil, Larynx and Paranasal Sinuses, Acta radiol. **15**:1, 1935.

21. Torrigiani, C., and Palumbo, V.: Radiumterapia dei tumori maligni delle prime vie respiratorie, Ztschr. f. Hals-, Nasen- u. Ohrenh. **40**:44, 1936.

22. Schintz, H. R., and Zuppinger, A.: Siebzehn Jahre Strahlentherapie der Krebse, Leipzig, Georg Thieme, 1937.

lesions of the piriform sinus; 7 of the patients had been free from symptoms for three years and 3 for five years. There were 14 lesions of the external arytenoid cartilage; 2 of the patients were free from disease for three years, and none were living after four years. There were 7 lesions of the epiglottis, with an average survival of twelve months. There were 19 lesions of the aryepiglottic folds, with 3 patients free from disease for three years and 2 for five years. Martin [23] reported on 4 intrinsic lesions, with 2 patients free from disease for two years or more, and on 53 extrinsic lesions, with 8 patients free from symptoms for two years or more. Torrigiani and Palumbo,[24] in a monograph published this year reported on 32 extrinsic lesions; in only 1 case did the patient survive three years. There were 25 lesions limited strictly to the vocal cord, all treated by the protracted fractional method; in 3 of the patients persistent infiltrative edema developed, becoming gradually worse. One had ulceration and necrosis, with sepsis; 5 died after one year with the disease, and 2 died after three years. Eight patients were living and free from disease at the time of publication, 2 for more than three years, 1 for five years, 1 for nine years and 4 still under observation, evidently in recent cases. Of 4 patients with subglottic lesions, 1 died in a year and 1 after three years; the fate of the other 2 was unknown. Of 11 patients with supraglottic but still intrinsic lesions, 1 had a primary regression but succumbed from recurrence; 1 died of cerebral hemorrhage; 2 were living after more than two years; 3 were reported to be still under observation, and the rest had succumbed. The authors admitted that the prognosis is bad for all but the strictly cordal lesions, since the supraglottic lesions tend to invade the pharynx and the subglottic tumors to invade the cartilage.

In the report on radium beam therapy research of the British Medical Research Council, issued in 1938,[25] of 8 patients with lesions classed as operable, 6 were free from symptoms, 3 for less than one year, 2 for more than one year and 1 for more than two years. Of the 16 patients with inoperable lesions only 1 was free from symptoms for less than one year. Peck and Maxwell [26] reported on 35 patients with inoperable

23. Martin, H. E.: The Treatment of Carcinoma of the Larynx, Tr. Am. Acad. Ophth. **41**:213, 1936.

24. Torrigiani, C., and Palumbo, V.: Curiethérapie des tumeurs malignes des voies aériennes et digestives supérieures, Bordeaux, les editions Delmas, 1940.

25. Medical Uses of Radium: Summary of Reports from Research Centres in 1937, Medical Research Council, Special Report Series, no. 232, London, His Majesty's Stationery Office, 1938.

26. Peck, W. S., and Maxwell, J. H.: Carcinoma of the Larynx: A Review of One Hundred and Seventy Consecutive Cases, Univ. Hosp. Bull. Ann Arbor **5**: 44 (June) 1939.

lesions treated by radiation, 4 of whom had survived for one-half year to two years without recurrence. Of 19 patients with advanced intrinsic and moderately advanced extrinsic lesions, 8 were alive and well for more than two years.

Reviewing these and similar statistics, one is impressed with the wide variance in the percentage of cures in similar groups of cases, which is in marked contradistinction to the uniform results of operation. Should this discrepancy be attributed to lack of uniformity in irradiation technic or to unpredictable factors as yet not understood?

In our own series I have tried to analyze the favorable cases for clues that might indicate the factors which brought about the successful outcome, but on comparison with similar cases in which unsuccessful results followed similar treatment, I find little on which to base conclusions. In a few cases the successful outcome might be logically explained; for instance, the case in which a small lesion on the vocal cord was removed for biopsy and was then irradiated. It is possible that in this case the lesion was completely extirpated by the biopsy forceps. I am quite sure that 1 of the cases reported by Imperatori was similar, and doubtless there have been others. In another case a lesion 0.5 by 1 cm., superficially ulcerated at the bifurcation of the cords and reported as squamous cell carcinoma, was irradiated for forty-one days, receiving a total of 188,000 milligram hours (13,800 r), with complete regression. Four years later a rough area appeared at the site of the original lesion, which aroused suspicion. A thyrotomy was done on the assumption that it was a recurrence. The specimen showed no evidence of malignancy, and the patient is alive and well seven years after the original treatment.

In another instance a man of 46, complaining of sore throat for three months and of dysphagia for three weeks showed a lesion on the left arytenoid cartilage and aryepiglottic fold, with fixation. Since there was some doubt as to whether the piriform fossa was invaded, the case was considered on the borderline, and irradiation was started, despite the fact that biopsy revealed a squamous cell rather than an anaplastic type of carcinoma, which is more common in this location. The patient received 100,000 milligram hours to each side (16,500 r), with the usual epidermitis. The lesion disappeared, and the patient was free from disease when last seen, two and one half years after the treatment. In view of so many failures in similar cases it is hard to explain the success in this one, in which the factors were all unfavorable, namely, fixation of probable infiltrative origin, a lesion spilling over the laryngeal introitus and an adult type of histologic structure of the tumor. Another case in which the factors were all unfavorable was that of a man of 58 with an ulcerated lesion 1.5 cm. in diameter in the left piriform fossa with fixation of the left cord but no palpable glands. Biopsy showed

a squamous cell carcinoma. He received a total of 172,000 milligram hours, eight to each side of the neck through 8 by 10 cm. portals at 10 cm. distance (28,000 r) over a period of forty-three days. There were some perichondritis and infection, resulting in an external discharging fistula and edema of the larynx. The symptoms subsided, and the last record was a notation two and one-half years later stating there was no evidence of disease and that the patient was at work. This is the only case of postirradiation necrosis that I have been able to find in which the patient recovered. On the other hand, a curious phenomenon which I have observed in 2 patients is the fact that when necrosis and fistulization have taken place as a result of the disease itself subsequent irradiation, instead of aggravating the condition, actually improves it. One of these patients, a man of 53, when first seen had an indurated mass involving the right side of the thyroid cartilage with a discharging fistula in the center. The tumor extended into the piriform sinus, involving the entire half of the larynx and the adjacent pharyngeal wall. Biopsy showed squamous cell carcinoma. He was given the usual protracted irradiation, which resulted in temporary arrest of the process and considerable relief. At least the irradiation seemed to control the secondary infection. Eventually, in the course of a year or so, the condition became worse, resulting in the patient's death. The other was a man of 60 who when first seen had a discharging fistula near the anterior border of the thyroid cartilage, surrounded by granulomatous masses which on biopsy revealed squamous cell carcinoma. The growth extended into the larynx near the anterior commissure and involved both cords. Surprisingly, the lesion yielded to irradiation, so that after one month the entire area, both external and internal, was smoothed over. While it is likely that this is only a palliative result, it seems to prove my point that irradiation carefully administered in the presence of spontaneous necrosis with external fistulization is an entirely different process from the mass chondro-osteitis that occurs as a result of irradiation. The former can at least be modified by irradiation, whereas the latter is practically hopeless.

Another point that I should like to bring out is the effect of irradiation on subsequent surgical treatment. It has been my experience that when operation has been attempted in a patient who has recently been subjected to intensive irradiation the chances for success are greatly diminished by reason of the previous treatment, and disaster is likely to follow. Certainly it is an item that must be taken into consideration by the patient when he decides to undergo irradiation therapy, particularly if it is a case in which a laryngectomy is under consideration. Coutard stated that in some instances it is difficult to determine whether fixation is due to infiltration or secondary to inflammatory reaction and

that the therapeutic test may be applied; namely, a moderate amount of irradiation will release the fixed cord if infiltration has not taken place. Should this fail, operation is indicated. For this purpose not more than 150 to 200 r should be given. Anything beyond this, however, when administered in the hope of arresting the growth with the reservation that if it fails operation may then be instituted, in my opinion only jeopardizes the patient's chances for recovery. I believe that it is difficult to tell in advance how much irradiation the tissues will tolerate before the circulation is so affected that healing after subsequent surgical treatment is delayed or may fail altogether. Let me cite a case in point. A 60 year old man with a squamous cell carcinoma involving the epiglottis, the arytenoid cartilage and the false cord was irradiated, with regression of the lesion. Three months later there developed symptoms of massive edema of the neck and intralaryngeal structures, indicating chondro-osteitis. The dyspnea made a tracheotomy a matter of urgency. The operation was followed by diffuse sloughing of the tissues and by sepsis, to which the patient succumbed. In another case a 49 year old man was treated for an intrinsic squamous cell lesion which failed to yield after he had received about half of the regular course of irradiation, at which time it was decided to do a laryngectomy. One week after the operation the wound broke open; diffuse sloughing ensued, followed by sepsis and hemorrhage, which caused the death of the patient. The lesson I learned from these cases was, first, that tracheotomy should be done as early as possible in the course of treatment, if not prior to its inception, and second, that patients submitting to irradiation in amounts intended to cure should be warned that if the lesion is operable or might later be considered so there is a greater risk than if the operation were done first and irradiation, if deemed necessary, reserved as a postoperative measure. Naturally I am aware of the fact that postirradiation surgical treatment has been successfully accomplished in many cases, and it is more than likely that the longer one waits to intervene the less is the danger. Yet when a patient undergoing irradiation therapy reaches a point where he or his physician feels that the treatment is not likely to effect a cure and a decision is made to resort to operation, it is not easy to sit back and wait three to six months, knowing that the lesion is steadily growing worse.

On the other hand, my observation of postoperative irradiation has been more favorable so far as tolerance is concerned. In the 9 cases whose records I have been permitted to examine there were no instances of deleterious effects from the irradiation, and 2 of the patients at present have been free from evidence of disease for one and three years respectively, a third patient having recently completed his treatment. I firmly believe that in borderline cases it is better to give postoperative

rather than preoperative irradiation, and I find a number of observers on record as of the same opinion.

And now we come to a brief discussion of the half-and-half method of treatment which has come into vogue in England and the Continent, meaning the Finzi-Harmer-Ledoux procedure with its various modifications. This procedure seems to have met with little favor in the United States, and, to my notion, logically so. The theory that by removing a block of thyroid cartilage without opening the larynx and irradiating the tumor through the window one avoids the danger of chondronecrosis and possible pulmonary complications while at the same time sparing the overlying soft tissues is in some respects fallacious. In the first place, infection is favored by reason of the foreign bodies (the radium tubes) buried within the tissues, and both Diggle [27] and Vogel [28] mentioned infection and necrosis as complications actually observed. It is surprising that the cartilage is not more frequently affected, since the bare margins of the windows are definitely exposed to the rays throughout the treatment, which means continuously for six to eight days. Second, the patient does not avoid an operation; on the contrary, he is subjected to two surgical procedures, the window operation with laying in of the radium tubes and the subsequent opening of the wound for removal of the tubes. Third, the postoperative course is prolonged by reason of local infection and reaction from the irradiation. The recovery period is much longer than that which follows laryngofissure. Lastly, the voice is no better in most instances than after laryngofissure.

Diggle [27] analyzed 26 cases of intrinsic lesions treated by this procedure between 1931 and 1935, in only half of which the diagnosis was substantiated by a biopsy. This fact detracts much from the value of the study, since the possibilities of error, especially with small lesions, are well known. Nevertheless, he recorded that 19 of the 26 patients are alive and free from disease for the following periods: one year, 1 patient; two years, 4 patients; three years, 9 patients; four years, 2 patients and five to six years, 3 patients. A good voice was obtained in 70 per cent of the cases. In comparing these figures with a series of lesions treated by laryngofissure he found a higher percentage of cures with the Finzi-Harmer method, principally because the statistics he consulted for operative results showed an 11 per cent postoperative mortality. In justice to the author I should mention that he called attention to New's and Jackson's published results following thyrotomy, in which there was no operative mortality. Since a great many set

27. Diggle, F. H.: The Treatment of Intrinsic Laryngeal Cancer, J. Laryng. & Otol. **52**:463 (July) 1937.

28. Vogel, K.: Erfahrungen der Berliner Universitäts Ohrenklinik mit Strahlenbehandlung bösartigen Geschwülste des Fachgebietes, Ztschr. f. Hals-, Nasen- u. Ohrenh. **43**:363, 1938.

their standards by the work of these surgeons, it is felt that laryngofissure should in properly selected cases be practically free from the danger of operative mortality. Therefore much of Diggle's argument is pointless. As for Vogel's [28] statistics, in which he claims as high as 90 per cent cures, I find it difficult to follow his arithmetic, since the best I can make out of his analysis is 40 of 56 patients free from disease for two to nine years, these being patients with strictly localized intrinsic lesions. In another series, of 36 intrinsic, and more extensive but still operable lesions, he reported 7, or 22 per cent, cured for three to four years. Even granting that the authors just cited have obtained cures of 70 per cent of unilateral localized cordal lesions, I still cannot see in what respect the method is preferable to laryngofissure, since it entails all the hardships of an operation plus the added discomfort of a prolonged convalescence, with no more assurance of obtaining a perfect voice. Certainly the few examples I have seen in my own locality failed to impress me, and so far as I have been able to discover no one in the United States has found it advantageous to follow this procedure to any great degree.

Before summing up this rather rambling discussion, I should like to make it clear that I have no quarrel with radiotherapeutists in general and no unreasonable antagonism to irradiation therapy. On the contrary, I have the greatest admiration for the many unselfish investigators who have labored in the hope of ameliorating human suffering. My contention is with those whose enthusiasm, to state the emotion euphemistically, sways their judgment in the selection of cases for irradiation, thereby depriving many patients of their best chances for cure by proved methods. I can find no evidence in the record that intrinsic lesions do better with irradiation than with laryngofissure or laryngectomy except when certain constitutional conditions exist as definite contraindications to operative treatment. These have been clearly set forth in the Jacksons' latest book. As for the extrinsic lesions which are beyond surgical intervention, one would have to determine from the extent of the lesion, the general condition of the patient and the histologic picture whether it is worth while taking the risk of full doses of gamma rays in the hope of a cure, assuming thereby the risk of chondritis, necrosis and the long convalescent period, or whether it is better to administer moderate amounts for palliation only. With continued experience and changes in technic it may be possible at some time in the future to carry a patient through a full course of irradiation without any danger of adding to his suffering, but at present the risk is still there and should not be minimized. As for the borderline lesions, I feel that, since postoperative irradiation is tolerated better than postirradiation surgical treatment, the patient should be operated on provided his general condition is satisfactory and then should be given an adequate course of irradiation,

since the results from irradiation alone are to date not sufficiently impressive to offer the patient any greater hope of cure than is offered by surgical intervention.

Finally, I should like to point out: 1. No patient should ever be subjected to either operation or irradiation without a preliminary biopsy. Jackson has emphasized this point over and over, yet I feel that it cannot be stressed too often. 2. The best results will be obtained only when the laryngologist, the radiotherapeutist and the pathologist cooperate to the fullest extent, without prejudice. The patient is entitled to the best that all physicians have to offer, and when a prognosis is given in a particular case it should be based on the combined experience of the best observers applied as closely as possible to the situation under consideration. Under no circumstances should a patient be misled by the wishful thinking of his physician, nor should he be permitted to make his decision without a full knowledge of what the available therapeutic agents have accomplished in the past.

HEAD NOISES

SIGNIFICANCE, MEASUREMENT AND IMPORTANCE IN DIAGNOSIS AND TREATMENT

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NEW YORK

In 1938 before the American Otological Society I reported some observations on the loudness of tinnitus and its role in accentuating deafness. Last year I reported on the masking of tinnitus. Cases were demonstrated by audiograms showing tinnitus in different frequency bands masked by pure tones at or slightly above the intensity of the tinnitus. Also demonstrated were cases in which it required 40 to 50 decibels above threshold hearing at the different frequencies to mask the tinnitus, and instances in which it was difficult and often impossible to mask the tinnitus with any applied frequencies or other masking noise. The explanation was offered that there are two kinds of tinnitus, vibratory and nonvibratory; that the first is easily masked; that the second is masked with difficulty, and that the ease of masking of nonvibratory tinnitus varies with the location of the lesions and with the accompanying deafness.

Further observations and the acquisition of a thermal noise apparatus [1] now enable me to report additional information and to venture further opinions regarding these measurements.

I have measured the loudness of the tinnitus by the loudness balance technic since 1927.[2] An approximate determination of the frequency of tinnitus has been attempted ever since a satisfactory audiometric method was introduced in 1922. With proper instruments it requires only a few minutes to obtain sufficient information for the loudness and frequency measurements.

The loudness balance technic I have described often. It is employed either monaurally or binaurally. The tone or band of frequencies of the tinnitus is determined by the application of frequencies first some distance above and below and then nearer and nearer to the patient's

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Read at the Forty-Sixth Annual Meeting of the American Laryngological, Rhinological and Otological Society, Inc., New York, June 6, 1940.

1. A thermal noise contains all frequencies in equal amounts. It sounds very much like air escaping under pressure from a nozzle, like the sibilant "sh."

2. Fowler, E. P.: Marked Deafened Areas in Normal Ears, Tr. Am. Otol. Soc. **18**:262-275, 1928.

estimation of the tone of his head noise, until he states that the same tone or about the same sound as his tinnitus has been attained. If the tinnitus is above 8000 it is often difficult to approach its frequency from the high tone side because of the universal uncertainty involved in judging very high frequencies. Below 8000 it is usually less difficult to judge even small differences in frequencies.

It is important to determine the intensity as well as the frequency of tinnitus. If the intensity of the applied sounds differs from the intensity of the tinnitus it will increase the difficulty in exactly locating the frequency of the tinnitus. The object is to duplicate both the intensity and the frequency (copy the head noises) as nearly as possible. It is better not to try to do this in the ear with tinnitus because of the certain presence therein of the recruitment phenomenon (due to the lesions causing the tinnitus, to the tinnitus itself or otherwise) and because this is usually the poorer ear. It is also the ear in which the neural elements occupied in producing and transmitting the tinnitus impulses are unavailable for sensing similar tones. Therefore, one should preferably balance the tinnitus in intensity and frequency with sounds applied to an opposite better, or even worse, ear if the latter suffers from obstructive deafness.[3] There will be less distortion and a more accurate estimate obtained in this way. Of course, there are several alternatives if this procedure is impossible or inadvisable.

I am not here primarily concerned with the effect of head noise as such, although this is important, but rather with its use as a diagnostic and prognostic agent. There are several characteristics which when present may suggest different places of origin, e. g., tonal character, broad or narrow frequency band, constancy, intermittency, reactions to certain physical influences and masking. The masking variations are measurable and therefore usable for correlation with other clinical findings.

The following points should be noted. If tinnitus is bilaterally similarly tonal and coincidental in timing and quality, it is difficult (but not impossible) to understand how it can originate only in the two peripheral mechanisms. It probably, then, is central in origin.[4] This does not mean that monaural tinnitus may not also be central in origin. If the tinnitus or applied sounds are bilaterally unequal in frequency or intensity they may seem to the patient to be in one ear only, because of a masking effect from the opposite ear. Such conditions may be substantiated by masking out the opposite ear.

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3. Obstructive deafness does not create the recruitment of loudness phenomenon.

4. The auditory neural mechanism is not unique in being subject to definite focal central lesions.

Auditory hallucinations may take on the form of various types of tinnitus. They result from cerebral irritative phenomena and may arise from any physiologic level of the neural apparatus of hearing—from lesions in the cochlea, cochlear nerve, ganglion cells, auditory tracts and protective pathways. They are not necessarily central, and have little or no dependable localizing value in diagnosis.

The spatial setting of tinnitus varies greatly. When tinnitus occurs with sharp dips or narrow deep troughs in the audiogram curve I have observed that its frequency occurs within the limits of the slopes. This was reported in 1928.[5] This, I suggested, indicated that both the tinnitus and the deafness arose from the limited lesions in the basilar membrane (or, in addition thereto, in the associated spiral ganglions). It seems improbable that such narrow gaps represent central lesions, because the latter, from the greater crowding of the neural elements, would produce wider areas of deafness and a more diffuse type of tinnitus. These observations contribute evidence that there is a close association in such lesions between the cause of the tinnitus and that of the deafness. The tinnitus occurs during the irritative phase of the lesion and foreshadows subsequent degeneration of the neural elements involved.

When tinnitus occurs with flat audiogram curves it is usually less localized in frequency. It sounds more like a roar, machinery, motors or waves; more like a broad band of frequencies, a thermal noise, etc. It is then not so apparent that the deafness and the tinnitus arise from the same lesions. I believe, however, that this is generally the case. There are exceptions to the foregoing general observations.

No one has reported hearing a tonal tinnitus or even a noise tinnitus by use of the cochlea microphonics or eighth nerve (Wever and Bray) responses.[6] Does this mean that the animal suffered no tinnitus? Does it not rather suggest that the lesions were too massive and that maybe the pick-up was not attempted at the appropriate time intervals after the experimental use of drugs, poisons, lesions of the middle ear or other measures. The more one studies the subject the more it appears that tinnitus is of central origin in a large proportion of instances.

Tinnitus represents more than just a subjective sound. It represents a reaction of the neural mechanism of hearing to certain extra-auditory

5. In this same paper attention was called to the fact that the tone of the tinnitus in a case of "narrow dip" was on the low tone side of the dip. It also was mentioned that in some instances the tone of the tinnitus is near the top of the curve on its high tone side.

6. Davis stated that "it would probably be impossible to distinguish the nerve impulses associated with tinnitus from the general background of physiological activity and the unavoidable noise level of our amplifiers."

or intra-auditory disturbances. In this respect it is similar to deafness. In certain stages it is suggested that it is a quantitative representation of the position and severity of such lesions. It is, in any case, important to find out everything possible about it.

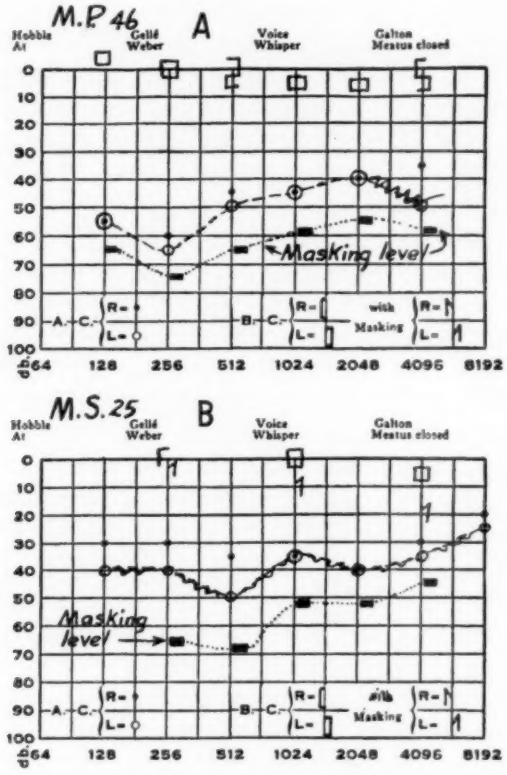

Chart 1.—Audiograms in 2 cases of otosclerosis. The patient in case *A* suffered from a pulsating and roaring, escaping air tinnitus in the left ear, of 10 to 15 decibels in loudness. This tinnitus was easily masked by frequencies of 10 to 15 decibels above the patient's threshold of hearing and by thermal noise. I believe that both the deafness and the tinnitus were of peripheral origin. Note that bone conduction was diminished only 5 decibels, unless it was originally high normal, in which case it may appear near normal even in the presence of a 15 to 20 decibel nerve deafness.

The patient in case *B* complained of a constant blurred whistling tinnitus in the region of 3000 in the left ear. This was felt as present even when not actually heard. This tinnitus was masked by 25 decibels over the threshold of hearing at 256 double vibrations and by progressively diminishing intensities at frequencies 512, 1024, 2048 and 4096. It was easily masked by thermal noise. The hearing improves when the tinnitus ceases and goes down about 10 decibels in the frequency area of the tinnitus when it is present. I believe that the deafness and the tinnitus are of peripheral origin. Note that bone conduction is increasingly lowered in the left ear with frequency, even though the reverse is true of air conduction.

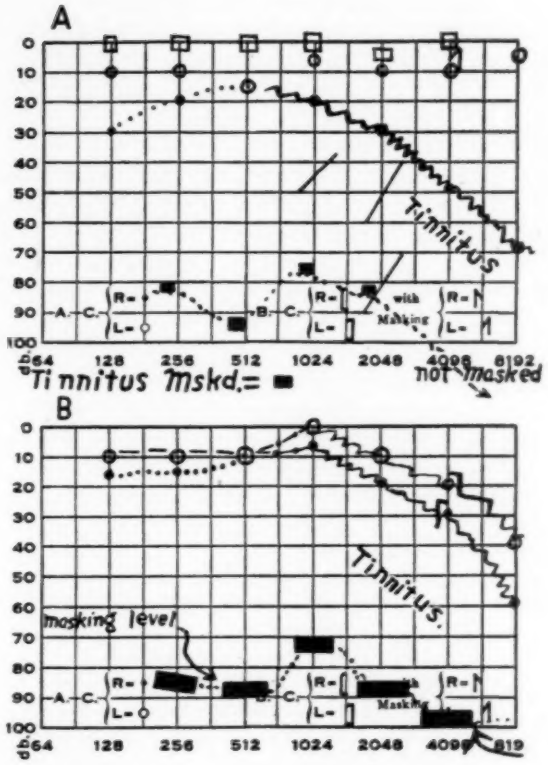

Chart 2.—Audiograms in 2 cases of healed otitis with evidence of scar tissue in the middle ear and a marked loss of hearing for the high tones in the worse ears. In both patients the tinnitus was high pitched and masked with difficulty at all frequencies; as shown in the graphs, it required from 80 to 90 decibels above the normal threshold to obliterate the sound of the tinnitus. It is my opinion that difficulty in masking may be due to the fact that the tinnitus is caused not by actual vibrations in the cochlea but by some biochemical change in the neural pathways. In order to eliminate the possibility of the tinnitus being heard also in the ear not being masked, binaural masking was also employed, with no change in the results. The patient in case *A* has hyperthyroidism. At the tinnitus frequencies it was impossible to mask the tinnitus. Masking noise in the opposite, normal ear also did not mask the tinnitus. At times, when the masking tones are very loud, this patient's tinnitus alternately appears and disappears.

The patient in case *B* had a history of gastrointestinal disturbances, bilateral tinnitus, vertigo for two years and no noticeable deafness. (Neither of these patients gave a history of otitis media, in spite of the evidence of scarred drums.) It may be that in both cases the difficult masking is due in part to the high tone deficiency (above 8000) in the masking apparatus, making it less efficient in masking very high tones. However, there is little or no tendency to increasing masking with frequency, which in my opinion, when present, indicates peripheral lesions. Moreover, apparently similar ears are commonly encountered wherein masking is accomplished at all frequencies at 10 to 20 decibels above the threshold. It would appear that in the 2 cases illustrated the lesions causing the tinnitus, in part at least, are central.

The neural impulses set up by both vibratory and nonvibratory autogenous excitations, like those set up by applied sounds, block other impulses from passing their place of origin and transmission in the neural mechanism. They therefore always raise the threshold for similar applied sounds. This blocking will vary according to the frequency and intensity of the mechanically produced tinnitus and in relation to the

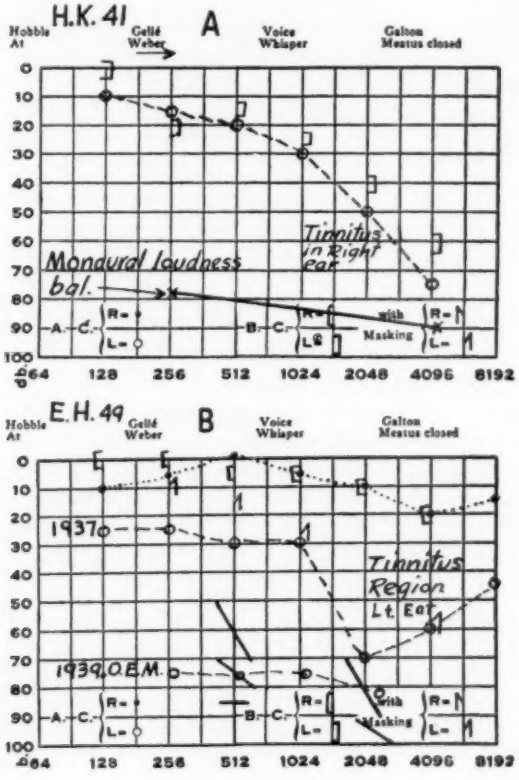

Chart 3

(See legend on opposite page)

extent and location of the lesions associated with nonmechanically (nonvibratory) produced tinnitus.

If tinnitus is mechanical in origin, its effects on the hearing will be exactly the same as those of similar applied sounds, because it is in fact an applied sound vibration which is sensed by the ear like any similar vibration. It is therefore easily masked by similar sounds slightly below it in frequnecy. When it ceases, no damage having been done, the hearing returns to normal.

If tinnitus is nonvibratory (biochemical) in origin, its effect on the hearing should show some differences from vibratory tinnitus or applied frequencies. Masking it should show variations dependent on the extent and position of the lesions which produced the tinnitus. Even though it ceases there may be little or no comparable change in the hearing, unless there is recovery (not merely cessation of excitation) in the neural mechanism involved. The more chronic the tinnitus the less probability of its cessation with a comparable gain in hearing.

There is no certain sign that a tinnitus is of peripheral vibratory origin, namely, the phenomenon of beats obtained by superimposing a

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EXPLANATION OF CHART 3

Chart 3.—*A*, total deafness in the right ear. There is possibly a remnant of hearing, but no words can be understood. The tinnitus in the totally deaf right ear is 20 decibels loud. It is masked only by the loudest buzzer or air noises applied simultaneously to both ears. The tinnitus in the left ear is very faint (5 decibels) and consists of a band of high frequencies. It also is masked with difficulty by discrete frequencies by either air conduction or bone conduction. Both middle ears appear normal. The air conduction and bone conduction in the left ear practically coincide, and both have gone down gradually during the past fifteen years. Thermal noise masked the left ear tinnitus at 80 decibels over normal thermal noise threshold. This is 5 to 40 decibels above the threshold level of the frequencies in the tinnitus region. In masking ears such as these, it must be remembered that sounds that are only 5 to 10 decibels above the high frequency thresholds are, because of the recruitment effect of nerve deafness, nearly or quite as loud as in a normal ear. Therefore, even 10 decibels over the threshold is very loud and will have a masking effect of at least 15 decibels and often up to 80 decibels as compared to the same sound in an ear without neural lesions. The progressive deafness with frequency and the normal hearing for low tones lead me to believe that the deafness and the tinnitus in the left ear are of peripheral neural origin. (By central neural is meant afferent to the end organ. By peripheral neural is meant within the end organ. By peripheral non-neural is meant vibrations instituted in or brought to the ear by actual mechanical means.)

B, the hearing in the right ear has always been near normal. In 1937 there appeared to be only 30 decibels of deafness in the left ear up to 1024, above which a marked valley or trough occurred in the threshold level, and both the binaural balance test and the bone conduction losses indicated at this time in this region a severe nerve deafness; however, the patient could not distinguish words spoken loudly into the ear. Two years later (1939) this ear appears to be totally deaf; no sounds of any kind are heard when the opposite ear is completely masked. The plotting on the chart is now only a shadow plotting, owing to inefficient masking. The tinnitus for the past two years has remained at about the same frequency band and is now heard only in the totally deaf left ear. Neither discrete frequencies nor broad bands of frequencies at loudest intensities masked the tinnitus. The tinnitus appears to be of neural origin. The deafness and the tinnitus are probably labyrinthine. Tinnitus referred to a totally deaf ear in itself suggests a neural origin (stump irritation). It certainly cannot be from peripheral vibratory stimulation. Both the auditory and the static labyrinths in the right and the left ear respectively in these 2 patients are now practically nonfunctioning.

proper applied frequency.[7] Central or peripheral nonvibratory tinnitus (see legend to chart 3) would not be expected to show this effect, because the lesions do not set up actual mechanical vibrations or sinusoidal stimulations. There are therefore no rhythmic comparable plus and minus phases to the stimulation of the biochemical irritations. Instead there is a mass stimulation of areas, which, however, because of some localizing or other spatial mechanism and because of a tendency in all stimulated neural elements to synchronize and take up a certain rhythm depending on their firing pattern, may be interpreted by the brain as of the same tonal quality or as a wider band of frequencies. (Davis stated the belief that "mere synchronization of impulses would not give a tonal quality to the resulting sensation.") The neural elements

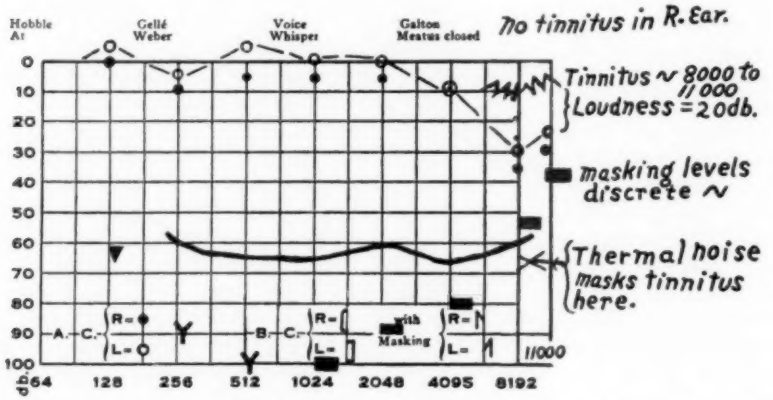

Chart 4.—The tinnitus in the left ear sounds like the high frequencies of thermal noise (like myriads of insects). The masking by discrete frequencies is close to the pattern obtained in masking applied sounds similar to the tinnitus. It occurred only above 512 and then progressively the closer it approached the frequency band of the tinnitus. The speech frequencies are little affected by the tinnitus, because the tinnitus frequency is above them. The bone conduction is normal for all frequencies up to 4096, above which, in most persons, it is not dependable, because it is heard less by way of the bone than by way of the air conducting mechanism. Thermal noise masked the tinnitus at 65 decibels over normal thermal noise threshold and 35 decibels over normal threshold at the tinnitus frequencies. It may be that the high intensity required for masking is due to a lack of sufficient loudness in the thermal noise frequencies above 9000. A frequency of 8192 masked the tinnitus at a setting of 55 decibels. This is 25 decibels over the threshold at 8192 and 5 decibels greater than the decibel loudness of the tinnitus. At 11000 masking occurred at 15 decibels over the threshold. There is at times a faint tinnitus in the right ear, similar in tone to that in the left ear. A mixed neural origin, peripheral and central, for the tinnitus with associated high tone deafness is suggested.

7. I have tried innumerable times to obtain beats by using my own tinnitus, but by the time I reached my soundproof room my tinnitus had ceased. I have even placed tuning forks near at hand, but without success. Their mere presence seems to prevent the appearance of my tinnitus.

are stimulated *"en masse barrage,"* each element firing with its own particular speed and intensity under the compulsion of the stimulant. There is no pattern of fundamental and overtone as from actual mechanical vibrations. This fundamental difference in setup is one of the causes for the variations in response to masking sounds. The masking sounds meet with a different pattern of resistance from both the "line busy" effect and the "line dead" effect.

It should be kept in mind that all of the neural elements at the site of a lesion are not necessarily implicated in causing audible tinnitus. Some (or many, or all) may be destroyed (degenerated, dead), and all such may be powerless alone to set up an audible irritation in the residue of active cells. Moreover, the lesions causing tinnitus are not necessarily confined within the frequency areas represented by the greatest deafness;

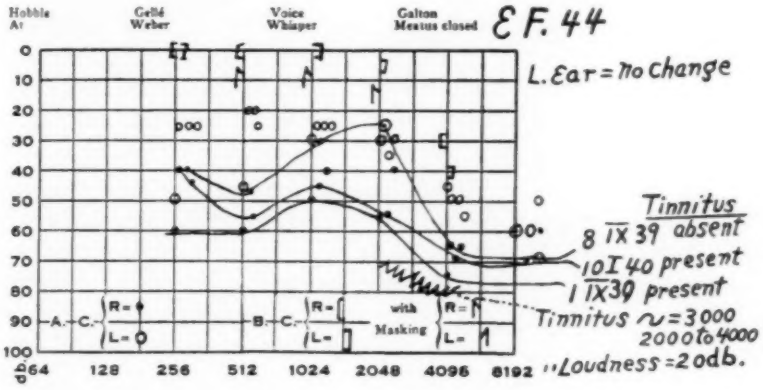

Chart 5.—Note the great variation in hearing at midfrequencies coincidental with changes in the loudness of the tinnitus and the small change at low and high frequencies. Only when the patient is overtired or tense do the menses increase tinnitus and diminish hearing in the right ear. Audiometric tests show no coincidental change in the left ear. Only the extreme swings in plotting for the right ear are shown by the continuous lines. The bone conduction at frequencies 1024 and 2048 varied 10 to 15 decibels, with twice these changes in air conduction. Thermal noise masked the tinnitus at 15 decibels above the normal thermal noise threshold. Although the tinnitus is in the region of 3000 (along the slope of the dip), the hearing change extends over several octaves below this frequency region. This is a frequent observation in several types of audiogram. It would appear that all parts of the lesion causing the deafness are not necessarily accountable for the tinnitus and that tinnitus and deafness do not necessarily correspond in frequency band or in degree. This tinnitus would appear to be neuroperipheral in origin.

not even confined within the tone areas the tinnitus represents, either on the basilar membrane or in the central auditory nerve distributions.

The place of greatest stimulation from severe biochemical irritations of tactile nerves is apt to be at the periphery of a lesion, at the zone

where the normal and the abnormal elements meet; in other words, in the area between normal sensitivity and lack of sensitivity, at the place of dominant irritation.[8]

The lesions causing nonvibratory tinnitus and the central effects of the nerve impulses set up by these lesions (the tinnitus) both affect the hearing: the first by putting out of business (dead wire effect) the auditory neural elements, and the second by transmitting and distributing the tinnitus impulses centrally through the neural paths and hence occupying these paths (busy line effect) for as long as the impulses are transmitted. The "busy line" and the "dead wire" effect are capable of study by use of appropriate masking methods.

It naturally occurred to me that one reason for the difficulty in masking very high tone tinnitus is that the masking tone was not sufficiently high or sufficiently intense. However, as this did not always occur, it was not the only reason for failure to mask. To eliminate this criticism, a thermal noise was used with very flat characteristics. It proved, as I have always contended it would, to be a most satisfactory masking noise, because it is made up of all frequencies from 128 to about 9000.

In the majority of instances tinnitus appears to be wholly or in great part within the frequency limits of this thermal noise. The illustrations demonstrate the variations found in masking tinnitus in comparison to the lack of variation found in masking applied frequencies. It would appear that when tinnitus is masked with unexpected difficulty it indicates a different causative mechanism from that observed with tinnitus that is easily masked. I believe it indicates not only that it is central in origin but that the deafness accompanying it also is of central origin to an extent measurable by the methods proposed.[9]

In many instances persons are told that nothing can be done for head noises and the accompanying loss of hearing and that they are doomed to suffer continued and even worse head noises and progressive deafness. Such a gloomy prognosis is avoidable when one has at one's

8. This is a common phenomenon in all tactile tissues; i. e., partial submersion in cold or hot water will give the greatest sensation of the cold or the heat at the level of the water on the body—the line where the more stimulated and the less stimulated areas meet, the zone where there are mingled the thermal extremes of contrast. The itching from severe burns and cutaneous ulcers occurs at the periphery, near the junction of the healthy and the diseased tissues, where there is the greatest mechanical irritation of the tactile end organs. Nonvibratory peripheral tinnitus may be thought of as a tickle or itch of the basilar membrane.

9. There is a deficiency in the number of neural elements, so that not enough are available to respond to the applied masking tone or noise to mask out the tinnitus.

command methods for management and medical and surgical treatment with which to control the tinnitus and also, in this way at least, to affect favorably one of the factors contributing to the patient's distress. Just because the head noise disappears and the hearing greatly improves (as it frequently does) one should not claim a "cure for deafness," even though the patient claims he has been restored to perfect hearing. Patients are prone to overestimate the hearing improvement when the noises cease, because of the relief from the annoying racket.

The close relationship of head noises and deafness is often not appreciated. Deafness may occur without head noises, but head noises cannot occur without deafness. Although not in itself a disease any more than deafness is a disease, tinnitus is one symptom of aural trouble and a danger signal of trouble ahead. Many treatments which are

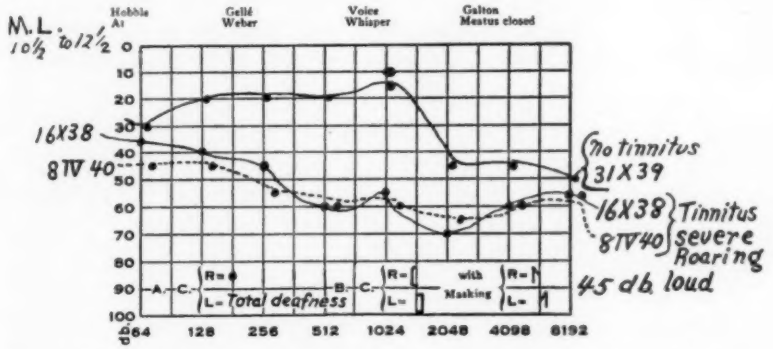

Chart 6.—Loud tinnitus (45 to 50 decibels) in the right ear, like a loud thermal noise. Total deafness and transitory loud tinnitus in the left ear, both following bilateral parotitis and meningitis four years ago, followed by vertigo and nausea for one to two months and marked loss in hearing in the right ear. When speech hearing in the right ear improves 40 decibels by air conduction and bone conduction at midfrequencies, the tinnitus in the right ear ceases. When the hearing goes down, the tinnitus returns. This happened several times. It is equivalent to an improvement in hearing from 1 foot (0.3 meter) to 20 feet (6 meters). Although one and a half years elapsed between the extreme low periods, there is little difference between these lows at the different frequencies. The bone conduction varied with the air conduction at all frequencies but when hearing was up, bone conduction at 128 was normal and at 250 was —10 decibels. No other changes were noted in the otologic picture. Thermal noise and air noise masked the tinnitus at 10 decibels over their thresholds. On account of the nerve deafness this is a very loud sound. By actual measurement it is equivalent to about 40 decibels in a normal ear. The patient thinks she "feels" the tinnitus in spite of the masking noise. Note that whether or not the tinnitus was present the hearing above 4000 varied little. These high frequencies are above those most important for speech, and therefore this lack of change did not prevent great improvement in speech hearing. All this suggests that the tinnitus is nonvibratory and that both the tinnitus and the variations in deafness are vasomotor in origin, both the peripheral and the central auditory neural apparatus being involved.

used as so-called "cures for deafness" appear to improve the hearing because of a coincidental alleviation of the head noises. This is often true of some operations, including fenestration of the semicircular canals, various operations on the mastoid and the middle ear and even removal of impacted cerumen and other substances from the external auditory meatus.

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COMPLICATIONS OF SURGICAL TREATMENT OF ACUTE MASTOIDITIS

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Medical statistics based on a sufficient number of patients to be of value must of necessity come from clinical centers, but the problem of the individual patient in the presence of complications of operation for acute mastoiditis has no such limitations. The otologist away from clinical centers, with less opportunity for counsel, confronted with more economic problems and with a limited personal experience, may have his surgical judgment put to the most severe test.

Otitic sepsis, thrombophlebitis, petrositis, labyrinthitis and meningitis are the major complications encountered. The incidence of and the measures for the relief of abscess of the brain following acute mastoiditis constitute an entity in themselves. A historical survey of the indications for surgical treatment in cases of acute mastoiditis shows a consistent trend to the position that, with some few exceptions, operation for acute mastoiditis is not considered an emergency procedure. The complications following surgical treatment of acute mastoiditis, meningitis excepted, have come to be placed in a similar category.

Brown [1] stated: "The general symptoms are the indications for surgery, and the local signs the guides for operative procedures."

With the advent of chemotherapeutic aids in the form of sulfanilamide and sulfapyridine, one is confronted with a preoperative complication in cases of acute mastoiditis. Use of these drugs was first reported in 1935; the literature has since become voluminous. Schenck [2] in 1938 published a splendid review of over two hundred and fifty titles. Incontrovertible evidence proves that the use of sulfanilamide for meningitis due to the beta hemolytic streptococcus is one of the great therapeutic advances of the past decade. Acknowledged to be markedly bacteriostatic and to possess unusual absorptive powers through parenteral administration, these drugs are not without power to cause toxic symp-

Read at a joint meeting of the Section on Radiology and the Section on Laryngology, Otology and Rhinology at the Annual Session of the American Medical Association, New York, June 14, 1940.

1. Brown, J. M.: Panel Discussion: Septic Thrombophlebitis of the Sigmoid Sinus; Surgical Treatment, Tr. Am. Acad. Ophth. **42**:457, 1937.

2. Schenck, H. P.: Use of Sulfanilamide in Otolaryngology, Arch. Otolaryng. **28**:698 (Nov.) 1938.

toms, such as weakness, tinnitus, dizziness, general malaise, anorexia, nausea, vomiting, acidosis and sulfhemoglobinemia. The theory of action of sulfanilamide and kindred drugs is not pertinent to this discussion and is not known. Suffice it to say that an interference in the assimilability of bacteria for proteolytic products (peptones, etc.) fits well into the clinical picture.[2a] These drugs may also interfere with the hemopoietic system, producing secondary anemia and granulocytopenia. They are capable of producing drug fever. Lesions in osseous tissue may be cut off from the circulation, and virulent organisms located in such lesions may, during the period of administration of the drug, retain their virulence without producing clinical symptoms.

With potentialities for both good and harm, but largely because of the masking effects, which lead to a sense of false security, administration of these drugs for acute otitis media with and without symptoms of mastoiditis is at present controversial.

Fisher [3] reported from the otolaryngologic department of Johns Hopkins University the use of sulfanilamide in 88 cases of otitis media due to the beta hemolytic streptococcus and compared these cases with 95 cases of otitis media due to the same organism but placed in a control group. In the former group there were but 7 mastoidectomies, with no complications, while in the latter there were 66 mastoidectomies, with 4 cases of septicemia and 1 death.

Galloway [4] reported from two hospitals in the same city. In 1937 at the Evanston Hospital, a private institution, there were 37 mastoidectomies, with 5 deaths, while in 1938 there were but 5 mastoidectomies with no deaths. During the same period at the Cook County Hospital there was no particular change in the relative occurrence of mastoidectomies.

Horan and French,[5] in an English naval hospital, reported the reduction of mastoiditis complicating otitis media from 23 per cent to 4.5 per cent with sulfanilamide therapy. The opportunity for early medication would seem to be a predominating factor.

Converse,[6] reporting from the Mosher Laboratory at the Massachusetts Eye and Ear Infirmary on 85 patients, concluded that sulf-

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2a. Lockwood, J. S., and Lynch, H. M.: Studies on the Mechanism of the Action of Sulfanilamide, J. A. M. A. **114**:935 (March 16) 1940.

3. Fisher, G. E.: Sulfanilamide in the Treatment of Otitis Media, J. A. M. A. **112**:2271 (June 23) 1939.

4. Galloway, T. C.: Chemotherapy, Arch. Otolaryng. **30**:901 (Dec.) 1939.

5. Horan, V. G., and French, S. G.: Sulfanilamide in Treatment of Acute Otitis Media, Brit. M. J. **2**:2519 (Dec. 31) 1939.

6. Converse, J. M.: Recurrence of Otitic Infections Due to Beta-Hemolytic Streptococcus Following Inadequate Sulfanilamide Therapy, J. A. M. A. **113**:1383 (Oct. 7) 1939.

anilamide therapy should be reserved for the treatment of spreading or life-endangering infections and should not be used for those of minor severity. These conclusions were based on the belief that the drug makes clinical evaluation more difficult because of possible toxic manifestations and recurrences after apparent recovery.

Maybaum, Snyder and Coleman,[7] from the otolaryngological service of the Mount Sinai Hospital, stated that the results of sulfanilamide therapy for meningitis are little short of marvelous but added their opinion that because of its masking effect its use should be reserved for otitic complications, such as meningitis, thrombophlebitis and abscess of the brain.

Hauser,[8] from the University of Pennsylvania, stated that the patients hospitalized there are not seen early in the course of otitis media and that in three years there was no material change in the incidence of acute surgical mastoiditis.

Rhoads and Afremow [9] reported a series of sore throats (31 cases) due to the hemolytic streptococcus, with a control group (36 cases) in which, according to their observations, sulfanilamide did not reduce the severity of symptoms, shorten the period of incapacity or reduce the incidence of complications or the duration of the carrier state.

This does not seem consistent with the general idea that this type of chemotherapy is most effective for diffuse lesions with maximal invasion and minimal destruction of tissues.

It is difficult for the individual otologist, striving to do his best for his patients, to reconcile reports from clinical centers that are practically contradictory. It would seem at times that such reports are predicated in the wish being father to the thought.

To condemn a therapeutic measure of acknowledged efficiency for calamitous complications for use in treatment of the minor conditions that lead to these serious complications does not seem logical. Rather, its usefulness should be acknowledged, with precautions against the potentialities for harm and a realization that temporary masking of symptoms does not mean final recovery. These considerations should permit use of the drugs to prevent complications.

The general practitioner and the pediatrician have adopted their use with enthusiasm, weighing an occasional abuse or drug complication against the benefits to the greatest number of patients.

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7. Maybaum, J. L.; Snyder, E. R., and Coleman, L. L.: Value of Sulfanilamide in Otogenous Infections, J. A. M. A. **112**:2589 (June 24) 1939.

8. Hauser, K. M.: A Survey of the Use of Sulfanilamide in Acute Otitis Media, Laryngoscope **44**:825 (Sept.) 1939.

9. Rhoads, P. S., and Afremow, M. L.: Sulfanilamide in the Treatment of Sore Throat Due to Hemolytic Streptococcus, J. A. M. A. **114**:942 (March 16) 1940.

Hauser [8] found by questionnaire that 89 per cent of physicians were using sulfanilamide for otitis media; 76 per cent stated the belief that it reduces the incidence of surgical mastoiditis, 8 per cent stated that it has no influence and 16 per cent were undecided.

Rather broad rules of procedure seem logical in private otologic practice. The earlier chemotherapy is used, the better the results. Hospitalization is desirable but not a necessity. Identification of the organism and frequent blood counts are a necessity. Determinations of blood concentration are preferred, but practicability and economy mean that the dosage will be determined by the clinical results. Rapid absorption and elimination call for doses at four hour intervals. Renal and hepatic complications demand extra precautions. A patient seen late, with a markedly increasing discharge, is not a candidate for chemotherapy. Marked clinical improvement should occur in seventy-two hours. With clinical and bacterial recovery, treatment with reduced doses should continue for ten days. The early discontinuance of treatment has been responsible for some of the adverse criticism.

Chemotherapy is not a panacea; it will fail at times; its use may be contraindicated; it may mask symptoms; it does not replace definitely indicated surgical intervention, but if it is used with judgment its good results at times are astounding and do not materially increase the hazard to the patient. This clinical interpretation may be termed a preventive complication of surgical treatment of acute disease of the mastoid.

Rather significant as to the time of operation in cases of otitic sepsis is a comparison of two reports. Lillie [10] reported a 3 per cent mortality from otitic septicemia occurring prior to operation and a 31 per cent mortality from the same condition appearing after operation, 13.2 per cent mortality for the entire group. Glass,[11] of England, reported a 57 per cent mortality from otitic septicemia, and the editorial comment [12] was the known fact that English surgeons operate earlier than do American surgeons.

Diploic and pneumatic mastoids present different problems. The structure of the mastoid cavity may combine the two. The diploic mastoid suffers essentially from osteomyelitis, and through thrombophlebitis of small venous radicles an almost concurrent otitic septicemia may be present. The pneumatic mastoid, being involved in osteitis, suffers from necrosis and breaking down of tissues, forming the coalescent mastoid,

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10. Lillie, H. I.: Suppuration of Temporal Bone Accompanied by Infection in the Blood Stream, Arch. Otolaryng. **18**:630 (Nov.) 1933.

11. Glass, E. J. G.: Blood Infection from Otitis Media, J. Laryng. & Otol. **48**:754, 1933.

12. Brown, E. V. L., and others: Year Book of the Eye, Ear, Nose and Throat, Chicago, The Year Book Publishers, Inc., 1934, p. 378.

and otitic sepsis occurs, largely by extension to and involvement of the larger venous channels through continuity of tissues. It is unusual for otitic sepsis beginning prior to operation to demand more than the simple or so-called complete mastoid operation as the first measure of surgical intervention. Certainly granulations found on the wall of a sinus, without other indications for intervention, should be left alone.

Continued sepsis after mastoidectomy occurs generally through involvement of the large venous channels, either as thrombophlebitis or as a venous thrombus. Diagnostic differentiation from clinical symptoms is not practical and not essential to the problem. The clinical picture may be confused, and there is considerable controversy as to the indications for treatment. Kopetzky, in his discussion of the paper by Brown,[1] stated that a sinus that pulsates always indicates thrombosis of the lateral sinus. Meltzer,[13] in a review, concluded that the appearance and feel of the sinus wall are not definite indications of the intravenous pathologic condition. The temperature and chill are not by themselves diagnostic. Tobey reported 11 of 78 patients to be without fever and 50 per cent without chills.[1] Ersner and Myers [14] reported elevation of temperature in 87.5 per cent and chills in 47.5 per cent of cases. The blood picture is not characteristic. The Schilling index is not of great diagnostic import, though the shifts have prognostic value. Hartmann, in discussing the paper of Brown,[1] stated that the presence of bacteremia is most important if qualified by the fact that positive blood cultures may be obtained early in any of the acute respiratory infections. Repeated positive blood cultures are of increasing diagnostic value.

While Eagleton stated that one positive blood culture is a definite indication for surgical intervention,[15] Meltzer [13] claimed that such a culture by itself is no indication for immediate operation.

Negative cultures give no assurance that operation should be postponed. Kopetzky [16] reported 17 such cultures in 28 proved cases of thrombophlebitis. Meltzer [13] reported 50 cases in which cultures were negative and in which 30 of the patients had thrombosis of the lateral sinus. Papilledema is of major significance. In the experience of Tobey it is present in 10 per cent, and in that of Dean, in from 25 to 33 per cent of cases.[1]

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13. Meltzer, P. E.: Treatment of Thrombosis of the Lateral Sinus, Arch. Otolaryng. **22**:131 (Aug.) 1935.

14. Ersner, W. S., and Myers, O.: Treatment of Thrombosis of the Lateral Sinus Without Ligation of Internal Jugular Vein, J. A. M. A. **109**:919 (Sept. 18) 1937.

15. Jackson, C., and Coates, G. M.: The Nose, Throat and Ear and Their Diseases, Philadelphia, W. B. Saunders Company, 1929.

16. Kopetzky, S. J.: Otologic Surgery, ed. 2, New York, Paul B. Hoeber, Inc., 1930.

The Tobey-Ayer test is of positive value if consideration is given to the fact that faulty interpretation may result from improper technic in making the test and to a realization of the anatomic variations of the jugular foramens as reported by Irish [17] and Mellinger (cited by Barnhill [18]). Obliterating thrombi are of slow growth and primarily protective. The venous flow is one of negative pressure, with practically complete anastomosis, so that circulatory disturbances seldom occur. Thrombophlebitis and thrombus have no doubt occurred many times, followed by recovery, without a diagnosis ever having been much more than suspected. Unsatisfactory convalescence, some sweating, slight chilliness and a pulse rate out of proportion to the temperature may be about all of the clinical evidence, which receives helpful confirmation in hemolysis of red cells and loss of hemoglobin. Blood transfusions as a means of immunologic therapy may offer something, but as a means of temporary aid to oxygen carriers which are debilitated and reduced in number they constitute a most valuable preoperative and postoperative therapeutic measure. Involvement of the mastoid cavity more often is entitled to clinical observation than to surgical treatment as an emergency. Likewise, in cases of suspected thrombophlebitis or thrombus, immediate operation may be postponed, either to prepare the patient as a better operative risk (Dean, cited by Brown [1]) or to obtain further clinical evidence to support the diagnosis.

There is not even complete accord as to indications for surgical treatment. Surgical intervention cannot promise complete obliteration of infection from the circulation, and certainly a completely collapsed jugular vein has performed an autoligation. Surveying the literature, one can go from Dixon,[19] who abstains from direct intervention, to Ersner and Myers,[14] who incise but do not disturb the thrombus, to Portman (cited by Kopetzky [20]), who has stated that in all cases of involvement of the sinus not only removal of the clot but ligation of the jugular vein is necessary. Meeting rather general approval is surgical intervention consisting of incision, obliteration of the sinus and removal of the clot by gentle suction (the clot being left at the bulb if not readily removed). Ligation is reserved as a secondary measure if clinical symptoms continue, except possibly in the presence of a mural thrombus

17. Irish, C. W.: Lateral Sinus Thrombosis, Ann. Otol., Rhin. & Laryng. **47**:78 (March) 1938.

18. Barnhill, J. F.: Surgical Anatomy of the Head and Neck, Baltimore, William Wood & Company, 1937, p. 82.

19. Dixon, O. J.: Newer Conceptions in Management of Septic Sinus Thrombosis, Laryngoscope **44**:448 (June) 1934.

20. Kopetzky, S. J.: Acute and Chronic Otitis Media, Sinus Thrombosis and Suppuration of the Petrous Pyramid, Arch. Otolaryng. **22**:336 (Sept.) 1935.

with marked sepsis and a positive blood culture. In these circumstances immediate ligation, as advocated by Almour and supported by Kopetzky,[20] is indicated.

The indications of surgical treatment of thrombophlebitis and thrombus, other than the local findings, depend not on any one clinical symptom but rather on an evaluation of the entire group of symptoms. Particularly is it to be emphasized that the formation of a thrombus is primarily a protective measure. In a coalescent mastoid, where continuity of tissue is a factor, the infecting organisms may lie deep within the thrombus. Waiting for confirmation, instead of immediate surgical attack on suspicion, receives general approbation. During the period of waiting, repeated blood transfusions, the use of specific serums and all supportive measures are in order. Again, chemotherapy has a most important place, and its use is to be guided essentially by the rules previously formulated.

Petrositis offers not only diagnostic difficulties but greatly increased technical problems of surgical approach. This is a complication that has to be met by the average otologist. The symptoms composing the clinical entity, postocular pain, late sudden increase in discharge and involvement of the sixth nerve, are frequently obscured. The evaluation of pain and otorrhea in their interrelations is important. Pain referable to the fifth nerve occurring late, when recovery has all but taken place, is of more significance than pain occurring immediately after the mastoidectomy. Involvement of the sixth nerve is significant, but absence of such involvement means nothing. The petrous bone varies in its structure, being diploic, pneumatic or sclerotic or representing a combination of types. The pathologic lesion, as in the mastoid, may be predominantly osteomyelitis, osteitis or a combination of the two. The information gained from roentgen examination may be invaluable but does not of itself indicate operative intervention. Just as in the mastoid cavity, it is often difficult to determine the difference between congestion and destruction of tissue. Definite cavitation to support clinical symptoms has greater positive value but is often not present. Petrositis no doubt occurs, and recovery takes place, without operative intervention. Involvement of perilabyrinthine areas and empyema of the apex have problematic differences.

Friesner and Druss [21] stated that suppurations in the petrous bone do not all extend to the apex and tend to drain and heal spontaneously. Myerson has stated that 80 per cent of patients recover spontaneously [22]

21. Friesner, I., and Druss, J. G.: Therapy of Suppuration of the Petrosal Pyramid, Ann. Otol., Rhin. & Laryng. **44**:1100 (Dec.) 1935.

22. Beck, J. C., and Guttman, M. R.: Treatment of Petrositis, Illinois M. J. **71**:333 (April) 1937.

and has urged conservatism. While petrositis may occur prior to or concomitantly with acute mastoiditis, in by far the greatest number of cases it becomes manifest after the mastoid operation.

With symptoms indicating possible involvement of the petrous bone, mastoidectomy is indicated. If this has been performed, a revision and careful search for a fistula or a diseased tract of cells should be made. True petrositis, inflammation and necrosis of tissue in a closed cavity, may not cause symptoms until the pathologic process encroaches on one of the walls. It is better to err on the side of conservatism. Failure to recognize indications for surgical treatment is not as culpable an error as subjecting the patient to the hazards of an operation on the petrous bone on the basis of insufficient symptoms and signs. The average otologic surgeon can follow a fistulous tract of a macroscopically diseased tract of cells with a probe and a curet, thereby possibly establishing an all-sufficient drainage. If an extrapetrosal pathologic condition is suspected in a case of postperilabyrinthine involvement, removal of the tegmen and following forward over the antrum and aditus may reveal a subdural abscess. Evident postlabyrinthine pathologic processes at a lower level may require removal of bone from the inner table of the sinus and Troutman's triangle to uncover a suspected subdural abscess in the posterior fossa. The question of performing a preliminary tympanomastoidectomy because of suspected apicitis or empyema of the apex is a real problem. Granting that the operation may be a life-saving measure and that the function of hearing is secondary, the preliminary tympanomastoidectomy, which requires extensive removal of the anterior wall, has not received universal endorsement. Williams [23] reported 30 cases of petrositis, in none of which was the tympanomastoidectomy performed.

The occurrence of a late postoperative increase in discharge from the middle ear would lead one to hope to find a fistula or a manifestly diseased tract of cells. In the absence of such leads and with the knowledge that every film showing a cloudy petrous bone does not mean coalescent petrositis or apicitis, the specialized technics of intrapetrosal approach to the apex of the petrous bone have such difficulties of detail that the average operating otologist had better be loath to accept the responsibilities involved. The extrapetrosal approach does not offer equal technical difficulties. Practically all types of operations (with the exception of Lempert's) are nothing more than drainage operations, and it would seem that the simpler technics outweigh the possible advantage obtained by the intrapetrosal approach with its position of drainage.

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23. Williams, H. L.: Petrositis, Proc. Staff Meet., Mayo Clin. **11**:493-495 (July 29) 1936.

Surgical attack on the perilabyrinthine area or the apex of the petrous bone is a formidable procedure, and, since chemotherapy constitutes the greatest advance in treating the one dreaded complication of petrositis, certainly this therapy is deserving of trial, particularly when manifest leads into the petrous bone are lacking. With improvement, continued application of the therapy is indicated even after subsidence of symptoms, in order to prove its efficiency over possible masking hazards. The fact that roentgen films taken in series attain their greatest value and that time is often necessary to establish regression of osseous pathologic change or establishment of true coalescent apicitis is an added recommendation for the use of chemotherapy.

Labyrinthitis occurring as a complication of acute mastoiditis does not constitute an indication for immediate operation. Goodyear [24] expressed the view that early operation for beginning labyrinthitis only hastens the spread of the infection.

Otitic meningitis, the most dreaded complication of acute mastoiditis, follows a general pattern, whether it occurs by direct extension from the middle ear or the mastoid cavity or follows otitic sepsis preceded by thrombophlebitis. The pathways of infection are important. The venous channels hold the position of first significance. Meningitis as an otitic complication is most common in early life, a period when the direct venous connections between the middle ear and the cranial vault are more evident. The symptoms are influenced largely by whether actual systemic involvement, limited meningeal irritation or increased intracranial pressure predominates. Chemical determinations of chloride content, increase of lactic acid, lowering of protein content and loss of reducing substances may be important adjuncts, but the presence or absence of an infecting organism, the increase or decrease in cell count and the spinal fluid pressure are the cardinal factors used to determine the pathologic process and the patient's status. The attempt to identify the infecting organism is the most important examination. Concomitant meningococcic meningitis can and does occur. The staining characteristics of degenerated organisms may vary, and only by culture may their real status be established (Eggston [25]). A spinal fluid which remains clear, with a high lymphocyte count, indicates probable syphilitic or tuberculous origin of the condition. Culture from loop implantation may fail and that from a centrifuged specimen be successful. Typing of pneumococci to determine the choice of specific serums now available is most important.

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24. Goodyear, H. M.: Indications for Surgery in Meningitis Secondary to Disease of Middle Ear and of Nasal Sinuses: Reservations in Early Surgical Intervention, Laryngoscope **49**:102 (Feb.) 1939.

25. Eggston, A. A.: Symposium on Otitic Meningitis: Diagnosis; Bacteriology of Suppurative Meningitis, Laryngoscope **49**:16 (Jan.) 1939.

Surgical drainage for meningitis has failed. Surgical treatment today is limited to the focus of infection.

Operation is indicated with appearance of the premonitory signs of meningitis. The proper evaluation of restlessness, irritabliity, headache, vomiting without nausea, pain in the neck without rigidity, photophobia, lagging of one upper eyelid and nystagmus is demanded.

Friesner and Rosenwasser [26] called attention to the fact that at times, as in cases of pneumonia, both otitis and meningitis may develop within a few hours, under which circumstances the otitis is simply an incident and not a focus of the meningitis.

In cases of diffuse suppurative meningitis the otologic surgeon is no longer justified in not immediately attacking an acutely inflamed mastoid cavity and following any leads that may be present. After operation, immediate revision of the mastoid cavity and search for leads is demanded; secondary foci may have become the important factors. It is in such circumstances that chemotherapy has achieved its great role. Such therapy should have the aid of everything possible, supportive and specific, blood transfusions and specific serums, repeated spinal taps and even intraspinal therapy; but the fact remains that until the advent of chemotherapy the prognosis of streptococcic and pneumococcic meningitis was practically nil.

CONCLUSIONS

1. Chemotherapy has changed the preoperative and postoperative pictures of acute mastoiditis.

2. Its potentialities for good outweigh its potentialities for harm. The latter are real, and contraindications to chemotherapy must be recognized. Possible masking is to be considered.

3. Indications for surgical treatment of the mastoid cavity, the lateral sinus and the petrous pyramid remain, but chemotherapy may prevent their development or cause regression so that operative intervention is unnecessary.

4. Sulfanilamide and sulfapyridine, the action of which is probably limited to the bacteriostatic effect, accomplish more the earlier they are used. They do not constitute a cure-all. Criticism based on the possible masking effect is unjust when therapy is not continued after the clinical symptoms subside. Therapy, barring contraindications, should continue for ten days after apparent clinical and bacteriologic recovery.

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26. Friesner, I., and Rosenwasser, H.: Symposium: What Is Desirable to Do in Otitic Meningitis? Surgical Treatment of Meningitis, Laryngoscope **48**:472 (July) 1938.

ABSTRACT OF DISCUSSION

DR. ISIDORE FRIESNER, New York: There are so many interesting points in Dr. Harkness' paper that it is obviously impossible to cover many of them in discussion. Furthermore, it is difficult to make a choice. One problem, however, presented indirectly by Dr. Harkness' paper is at present of prime importance to the otologist.

On the one hand, there are such reports as Fisher's, of Johns Hopkins University, which shows approximately an equal number of patients with otitis treated chemically and of untreated controls. In the latter group there were almost ten times as many mastoidectomies as were performed on patients treated with sulfanilamide. On the other hand, there is a report by Converse, of the Mosher Laboratory, to the effect that sulfanilamide therapy should be reserved for the treatment of spreading or life-endangering infections and not used for mild infections. These conclusions were based on the belief that the drug makes clinical evaluation more difficult.

Masked mastoiditis presents no new problem, but rather an old one that is recurring with far greater frequency under chemotherapy. With the natural enthusiasm for the drug on the part of the internist and pediatrist based on such reports as Fisher's, the difficulty that otologists are facing is the question of their attitude toward the drug, which should counterbalance the enthusiasm of the physicians. Perhaps the report of Fisher and that of Converse are the extremes.

For a long time it has seemed to me that otitic infections divide themselves into two main groups. There is, first, simple empyema of the middle ear. Second, there seems to be a group of conditions which, for some reason that is not understood, rapidly go on to what might be termed panosteitis of the temporal bone.

While chemotherapy can and does seem to have a curative influence on the first group, it does not seem to cure the second group. It is with the latter that the masking effects of sulfanilamide are particularly dangerous. Perhaps the rapid spread of the infection by Streptococcus haemolyticus beta is, to a considerable extent, due to its fibrinolytic property.

In an attempt to clarify this problem in my own mind, I recently asked Dr. Gregory Shwartzman, bacteriologist at the Mount Sinai Hospital, whether sulfanilamide influences the fibrinolytic character of the streptococcus.

Experimental proof at the hospital is to the effect that it does not do so. "It kills the organisms," says Dr. Shwartzman, "when they are amenable to body fluids containing an adequate concentration of the drug. But it does not influence the biologic characteristics of the streptococcus."

Perhaps in cases of panosteitis the streptococci are not amenable to the influences of sulfanilamide. This may be because the infection in the submucosa rapidly causes infectious thrombi in the vessels, which are not reached by the drug.

In a recent communication before the American Otological Society, Dr. Champ Lyons expressed the belief that chemotherapy should have its effect in forty-eight or seventy-two hours. It therefore seems rational that, as it is known that chemotherapy has its maximum effect if used early in the course of infection, the otologist should set a time limit for the use of this drug of approximately six or seven days from the onset of the infection and then observe any phenomena suggestive of osseous disease when chemotherapy has been abandoned.

It might be asked: What is the basis for the belief that in the presence of signs suggestive of masked osseous disease sulfanilamide therapy should not be continued vigorously? I can only cite 2 cases in which patients with otitis of one or two days' duration were admitted to the hospital with meningitis due to Str.

haemolyticus beta. They were treated energetically with sulfanilamide. They recovered from their meningitis and yet, despite the vigorous chemotherapy, had suppurative mastoiditis which required operation.

Dr. Frederick M. Law, New York: I have to speak from the standpoint of a roentgenologist. I agree with everything which has been said, but there are two observations which I should like to make.

Sulfanilamide is a valuable but treacherous drug. When it is administered, the condition must be followed by a series of roentgen films in order to obtain knowledge of what is taking place within the mastoid process. When the drug is given early, before any involvement of the bone occurs, the prognosis is good. If it is administered after beginning osseous change, however, the symptoms are relieved and the patient "feels fine," but the drug has no effect on the bone other than delayed action of the process.

When administration of the drug is discontinued, the osseous destruction proceeds just as though no drug had been administered, and it is absolutely necessary to use follow-up films, which should be stereoscopic, as was stressed by Johnson.

I have had reports that operation after administration of sulfanilamide revealed complete destruction of the bony walls, whereas the roentgen ray showed cell structure present. The reason for that is that the films were not made stereoscopically to obtain the third dimension, and the apparent cell structure shown on a single film was caused by a layer of complete and incomplete cells immediately under the cortex.

Visualization in the third dimension would have shown that the deeper cells were destroyed. The cell structure would have no depth. I feel that one must have absolute faith in the films in order to know the progress of the disease. It is known that resolution occurs in the presence of osseous necrosis, but it is safer to be aware of the condition than to feel comfortable in the absence of all symptoms.

The other condition I should like to mention is petrositis. It has been thoroughly described. Marked involvement of the petrous bone can be shown on a roentgenogram, but I should like to caution against interpretation with only a single set of films. One should be made at the beginning of mastoiditis for comparison in case of future trouble.

The petrous bone is just as susceptible to anatomic variations as is any other bone of the skull, and I have seen variations in the structure simulating a petrositis with absorption of the petrous bone. If one observes films taken for a regular sinal involvement one will find many times an anatomic variation, which must be considered in analyzing the appearance in the presence of positive symptoms.

Dr. Gordon F. Harkness, Davenport, Iowa: The purpose of this paper was a plea to place chemotherapy on a rational basis. I was very glad to have Dr. Friesner put a time limit on the administration of these drugs. In my paper I said seventy-two hours. If marked clinical improvement does not occur, administration should be stopped. Also, I think the point that Dr. Law made, concerning the follow-up with roentgenograms during chemotherapy, is most important.

OPERATIVE TREATMENT OF OTOSCLEROSIS

REPLY TO A RECENT CRITICISM

GEORGE E. SHAMBAUGH JR., M.D.
CHICAGO

Frank and open criticism of any new form of therapy is not only desirable but necessary before an evaluation of the therapy can be arrived at by members of the medical profession. For this reason physicians who treat otosclerosis surgically were particularly interested in the recent article by L. K. and P. Guggenheim [1] entitled "Artificial Fistula in Cases of Otosclerosis." Unfortunately, the authors' only experience with this kind of surgery was the observation of a single case in which an unsuccessful operation was performed by some one else plus some experiments on the regeneration of bone in the rabbit's skull (not on the labyrinthine capsule). As a consequence of their restricted experience, the authors were led to make certain statements which should not be allowed to go uncorrected.

The authors stated:

> The treatment of otosclerosis by means of (1) reversal of halisteresis and (2) regeneration of the damaged neural mechanism through dietary regulation and the administration of calcium, phosphorus and vitamins has after three years of experimentation succeeded in producing improvement in hearing far greater than has any fistula operation thus far reported.

To my knowledge, which includes a complete survey of the world literature carried out each year in preparing the annual summary of the literature on otosclerosis for the ARCHIVES OF OTOLARYNGOLOGY, there have been no audiometrically substantiated improvements in hearing in cases of otosclerosis from any other treatment, medical or surgical, as great as those reported following the Lempert operation. The authors have not presented in any of their writings audiometric substantiation of their claim of improvement in hearing from dietary measures, and yet I am sure that they will agree that no treatment for otosclerosis can be accepted as of proved value until it is backed up by accurate hearing tests. All critical observers agree that deafened persons are suggestible and that stimulated by hope (induced by almost any form of treatment) they begin to pay greater attention and therefore seem to hear better. This apparent improvement, however, is psychologic and not physiologic; it

1. Guggenheim, L. K., and Guggenheim, P.: Artificial Fistula in Cases of Otosclerosis, Arch. Otolaryng. **32**:1 (July) 1940.

cannot be substantiated by accurate hearing tests, and it never lasts. Since the audiometer is the most accurate method available for measuring hearing, since the audiometric curve sometimes improves just with practice, and since the hearing level in cases of otosclerosis normally fluctuates within a range of 10 decibles, the authors will agree that no treatment for deafness can be accepted as of proved value unless repeated audiograms before and after treatment show a maintained improvement in hearing at least 10 decibels above the pretreatment level. Finally, to be of practical value this improvement must include the conversational frequencies (512, 1024, 2048).

I am looking forward to audiometric proof of the value of the authors' dietary treatment of otosclerosis. Meanwhile I call their attention to the fact that Lempert's published audiograms are the first acceptable proof ever to be offered of significant and lasting improvement in hearing in otosclerosis from any form of treatment. Further, I can substantiate Lempert's results by my own experience with the fenestration operation. From July 15, 1938 to the date of writing (Sept. 20, 1940) I have surgically treated a total of 31 patients with otosclerosis, using the Lempert one stage fenestration technic. The results follow. (The hearing level in each case is expressed as average decibel loss for the conversational frequencies. The preoperative hearing level is the average of two to nine audiograms, while the postoperative hearing is the most recent audiogram. If one wishes, the average decibel loss or gain in each case can be converted quickly into percentage loss or gain by multiplying the value given in decibels by 0.8.)

CASE 1

July 13, 1938: Fenestration of left ear. Fistula closed in six weeks and hearing gain lost.

Aug. 16, 1939: Fenestration of right ear. Fistula closed in six weeks and hearing gain lost.

Jan. 24, 1940: Revision of left ear. Fistula has remained open, with sustained hearing gain averaging 37 decibels above the preoperative level for the conversational frequencies.

CASE 2

July 20, 1938: Fenestration of right ear. Membranous labyrinth ruptured. Slight improvement that did not last more than two months.

CASE 3

July 27, 1938: Fenestration of right ear. Postoperative serous labyrinthitis, with further loss of 5 decibels for conversational tones in ear operated on (one with poorer hearing).

CASE 4

Aug. 17, 1938: Fenestration of right ear. Fistula closed in three weeks and hearing gain lost.

CASE 5

Oct. 12, 1938: Fenestration of right ear. Membranous labyrinth ruptured. No improvement.

Sept. 11, 1940: Revision of right ear. Fenestrum made in superior canal. Hearing improved; at time of writing 8 decibels above original preoperative level.

CASE 6

Oct. 26, 1938: Fenestration of right ear. Fistula closed in six weeks and hearing gain lost.

CASE 7

Nov. 9, 1938: Fenestration of left ear. Fistula closed in five weeks, with loss of hearing gain.

Jan. 2, 1939: Fenestration of right ear. Fistula partly closed, with partial loss of gain.

April 23, 1940: Revision of right ear. Fistula has remained open, with hearing gain 30 decibels.

CASE 8

July 13, 1939: Fenestration of right ear. Fistula open, with sustained hearing gain 14 decibels.

July 17, 1940: Fenestration of left ear. Fistula open, with hearing gain 29 decibels.

CASE 9

Aug. 9, 1939: Fenestration of right ear. Fistula partly closed in three weeks, with loss of hearing gain.

July 31, 1940: Revision of right ear. Fistula open, with hearing gain 14 decibels.

CASE 10

Nov. 2, 1939: Fenestration of right ear. Fistula partly closed in six weeks, with loss of hearing gain.

CASE 11

Nov. 22, 1939: Fenestration of left ear. Fistula open, with hearing gain 13 decibels.

CASE 12

Jan. 4, 1940: Fenestration of right ear. Fistula open, with hearing gain 14 decibels.

CASE 13

Jan. 17, 1940: Fenestration of right ear. Fistula partly closed, with loss of hearing gain.

May 15: Revision of right ear. Fistula open, with hearing gain 21 decibels.

CASE 14

Feb. 7, 1940: Fenestration of right ear. Fistula open, with hearing gain 24 decibels.

CASE 15

Feb. 21, 1940: Fenestration of left ear. Fistula open, with hearing gain 11 decibels.

CASE 16

Feb. 28, 1940: Fenestration of left ear. Fistula partly closed, with loss of hearing gain.

July 25: Revision of left ear. Fistula open, with hearing gain 28 decibels.

CASE 17

March 13, 1940: Fenestration of right ear. Fistula open, with hearing gain 34 decibels.

CASE 18

April 24, 1940: Fenestration of left ear. Fistula open, with hearing gain 11 decibels.

CASE 19

May 24, 1940: Fenestration of left ear. Fistula open, with hearing gain 25 decibels.

CASE 20

June 19, 1940: Fenestration of right ear. Fistula open, with hearing gain 27 decibels.

CASE 21

July 10, 1940: Fenestration of right ear. Fistula open, with hearing gain 14 decibels.

CASE 22

July 11, 1940: Fenestration of left ear. Fistula open, with hearing gain 21 decibels.

CASE 23

July 18, 1940: Fenestration of left ear. Fistula open, with hearing gain 36 decibels.

CASE 24

July 24, 1940: Fenestration of left ear. Fistula open, with hearing gain 21 decibels.

CASE 25

Aug. 1, 1940: Fenestration of right ear. Fistula open, with hearing gain 19 decibels.

CASE 26

Aug. 7, 1940: Fenestration of right ear. Fistula open, with hearing gain 27 decibels.

CASE 27

Aug. 14, 1940: Fenestration of left ear. Fistula open, with hearing gain 8 decibels.

CASE 28

Aug. 21, 1940: Fenestration of right ear. Fistula open, with hearing gain 32 decibels.

CASE 29

Aug. 28, 1940: Fenestration of left ear. Fistula open, with hearing gain 28 decibels.

CASE 30

Sept. 4, 1940: Fenestration of left ear. Fistula open, with hearing gain 24 decibels.

CASE 31

Sept. 5, 1940: Fenestration of right ear. Fistula open, with hearing gain 25 decibels.

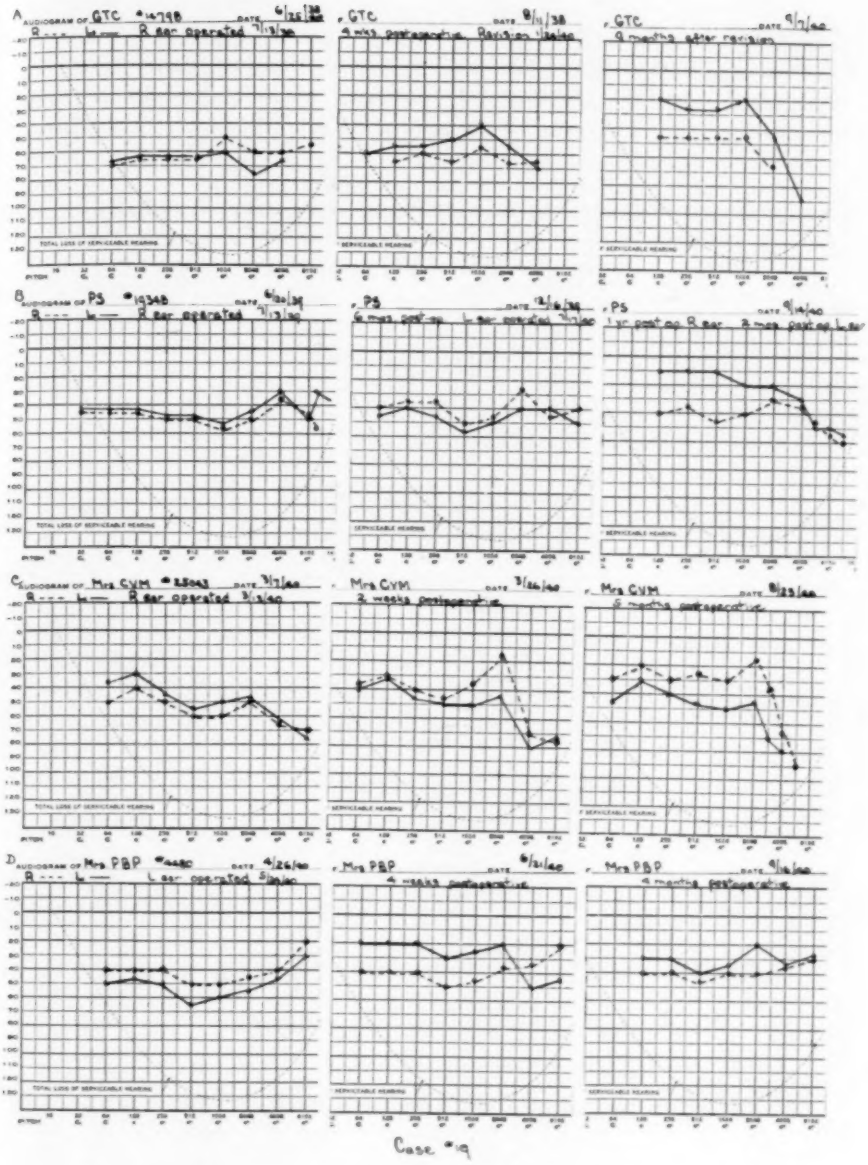
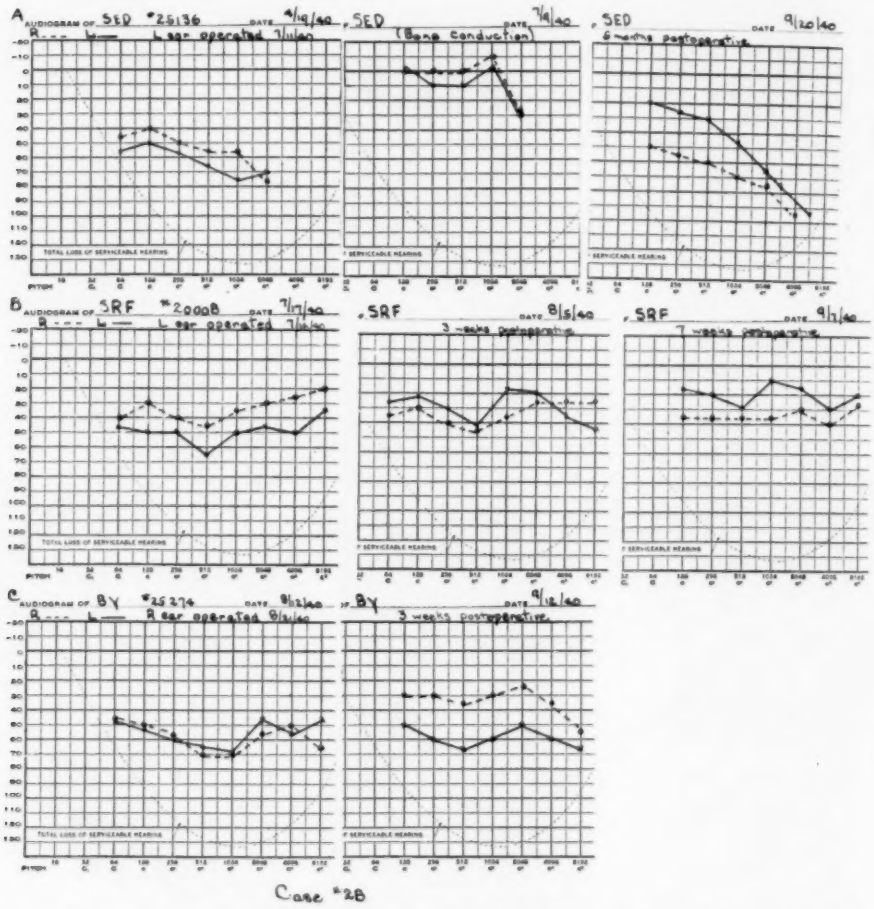


Fig. 1.—Audiograms: *A*, case 1. *B*, case 8. *C*, case 17. *D*, case 19.

Summarized, my results show that of 31 patients operated on, 24 now have an improvement in hearing, audiometrically substantiated, of more than 10 decibels for the conversational frequencies and therefore of

practical value to the patient. This means that a practical improvement in hearing has been secured in 77 per cent of all the patients operated on from July 1938 to September 1940. The gain in hearing varies from 11 decibels (case 15) to 37 decibels (case 1) for the conversational tones. While it is true that 1 or 2 of my most recently treated patients might yet lose their improvement by closure of the fistula, it is also true that



Case #28

Fig. 2.—Audiograms: *A*, case 22. Note loss of bone conduction for a tone of 2048 double vibrations and failure to recover major part of hearing for this tone. *B*, case 23. *C*, case 28.

these most recent ones have not yet reached the peak of improvement in their hearing. Of the last 26 consecutive patients operated on, from Nov. 21, 1939 to Sept. 21, 1940, improvement in hearing has been secured in every case. This group includes 6 patients in whom the

original fistula had closed entirely or in part with loss of the original gain and on whom revisions were performed.

When comparable improvements in hearing in a similar percentage of patients treated have been reported following dietary management or any other type of medical or surgical treatment, I shall adopt that method in place of the arduous fenestration technic.

On page 3 of their article Guggenheim and Guggenheim stated:

> The ingenious, difficult and time-consuming technic which has produced coast to coast enthusiasm has nothing whatever to do with the success or failure of the fistula operation. . . . There is little doubt that the old and simple approach to the horizontal canal posteriorly and covering of the fistula with any membrane at hand will work as well and take one sixth of the time.

As no one has yet reported any audiometrically proved results with a simplified technic and since my own failures were nearly all the result of errors or omissions in the "ingenious, difficult and time-consuming technic," whereas my best results were in direct proportion to the precision and exactitude with which each step in the "ingenious, difficult and time-consuming technic" was accomplished, I must dispute the authors' statement. I must also deprecate it as dangerously misleading and as having no basis in fact either in the authors' own experience or in the published experience of others.

The final sentence in the article, "No mention has been made of the approach through the canal as opposed to the posterior approach as the time-honored posterior one is obviously superior for all purposes," would be justified only if the authors had themselves tried the endaural approach and were therefore in a position to reach an opinion.

In conclusion, I invite and urge Drs. Guggenheim to see and examine any or all of my patients, to test the hearing of any patients before as well as after operation and then, when they are in a position to render a judgment, to publish their opinion of the results as they have observed them.

122 South Michigan Avenue.

USE OF ANTICOAGULANTS IN CASES OF POST-OPERATIVE THROMBOSIS AND EMBOLISM

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For a general surgeon to present the problem of thrombosis and embolism to an otolaryngologic society it will be necessary to draw comparisons between etiologic factors in the extremities and in the skull.

After discussing the subject with a number of otolaryngologists, I feel that there are certain operations in which the general principles of etiology and treatment involved are similar. Before discussing the therapeutic problem it seems advisable to review and classify the clinical types encountered. There are, roughly, four main types of thrombophlebitis. While the mean of each type is a distinct entity at the extremes, there are gradations from one type to another. Their classification is as follows:

1. Thrombosis. This is the type most commonly encountered in general surgical practice. A silent thrombus forms in the extremity, and suddenly, without warning, an embolus is broken off and transported to the lungs, sometimes causing immediate death. In this particular type the thrombus is not particularly septic; there is little periphlebitis; the temperature is not elevated, and the patient has not complained in general of tenderness in the leg. There may be no edema.

2. Thrombophlebitis. Thrombophlebitis is associated with a clot in the vein and periphlebitis. All physicians are conversant with this type, which in the lower extremity produces swelling and tenderness, with usually a sudden rise of temperature. The condition usually persists for seven to eight days and then gradually subsides. Embolism is not as common with this type as with that first mentioned, but in the series analyzed in Gallie's clinic in Toronto, Canada, 15 per cent of such conditions produced embolic phenomena.

3. Suppurative thrombophlebitis. This is a more septic condition than those previously described. It is more often noted with diseases of the mastoid and with processes arising from carbuncles of the upper lip and of the nares. It is occasionally seen in general surgical practice.

Read at the Forty-Sixth Annual Meeting of the American Laryngological, Rhinological and Otological Association, Inc., New York, June 7, 1940.

4. Periphlebitis. This is more apt to be a migratory type of condition; it is often not associated with a clot within the lumen, and the main pathologic condition is found in the lymphatics traversing the adventitia of the vein. This is associated clinically with tenderness and redness of the skin and subcutaneous tissues surrounding the vein, elevation of temperature and often continued sepsis. It is rarely associated with embolic phenomena.

It is generally accepted that dehydration, venostasis, trauma and infection are the main factors that initiate the collection of platelets on the intima of the vein, which is followed by laking of blood cells and formation of fibrin to create the thrombus. If the thrombus did not propagate but remained a local occlusion, there would be little cause for worry. There might be local tenderness and distal edema, which later would disappear as a collateral blood supply developed. But with the propagation of either a waving red or a septic thrombus which can at any moment break off one is intensely concerned.

If I am correct in interpreting the types of thrombophlebitis encountered in otolaryngologic practice, they may roughly be divided into two main groups—first, those arising from the middle ear and the mastoid, where there is an abscess within the bony cavity in close proximity to a neighboring vein. The vein becomes inflamed by contiguity, and because there is no opportunity for dilation, as the vein is encompassed within bony canals, thrombosis occurs, and the process extends by continuity from the smaller venous sinuses until it reaches the jugular vein. General surgeons are accustomed to see a similar process occur in osteomyelitis of the long bones; the initial process starts in the metaphysis of a long bone, and retrograde thrombosis of the nutrient artery results, ending in sequestration of the involved shaft. The second most usual cause, I believe, is carbuncle of the upper lip or of the nares, which results in thrombophlebitis of the angular vein and an extension from this process onto the floor of the skull.

Homans has shown that emboli are most likely to break off when the process extends from a smaller vein into a larger one and eddies are created. Thus, in the leg a common origin of thrombosis is in the larger veins of the calf, beneath the gastrocnemius muscles, and in the junction at Scarpa's triangle of several veins in the thigh. May there not be a similar connection in the skull and neck? The otolaryngologist, then, is not so much concerned with a silent and relatively aseptic thrombosis as with the infection which produces the septic emboli and, as a sequel, meningitis, abscess of the brain or distal embolic phenomena.

Fortunately for the otolaryngologist, motion does not occur within the skull, so that emboli are not as easily broken off, and gravity aids in drainage of the veins. The general surgeon's problem is concerned

with dependent drainage in the veins of the leg, where gravity works against the patient, with the danger of muscular effort detaching the embolus.

THERAPEUTIC MEASURES

Granting that sepsis, venostasis and dehydration are the main factors in producing thrombophlebitis, there is probably another factor which is inherent in the patient. In 1928 Stanley-Brown and I started a study of thrombosis and embolism and directed our attention to determining whether we could determine from examining a patient's blood whether he was susceptible to intravascular clotting. We believe that in all fields of medicine one is concerned with hyperactivity and hypoactivity, such as hyperthyroidism and hypothyroidism or hyperemia and anemia. Granting that there are potential bleeders, we felt that there might readily be potential clotters. We started our studies with an analysis of prothrombin, fibrinogen and antithrombin. During ten years we examined the blood of well over 8,000 patients. This examination consisted in routine anteoperative examination, postoperative examination of normal patients and examination of patients who had had either thrombophlebitis or embolism. We found that almost without exception, patients who had vascular accidents had increased clotting factors. Starting on a concept that we might be able to build up a prophylactic regimen for patients who after operation had high clotting factors, we presented a diet low in fats and proteins and, after advice from Dr. Loeb of Columbia University, began intravenous injection of sodium thiosulfate. As far as we are able to determine, sodium thiosulfate probably acted by aiding the liver to liberate heparin. In 1936 we started with heparin but found that its action was so short that the anticoagulant effect disappeared within an hour after the injection. Also, at that time heparin had not been purified as it has been today, and many toxic results followed its use.

Heparin was originally discovered by Howell. It is considered to be a mucoitin polysulfuric acid and is obtained in the laboratories now by using large amounts of animals' lungs. It may well be the substance which in previous physiologic studies was called antithrombin. It is unquestionably an excellent anticoagulant. The rationale of its use as a prophylactic therapeutic measure is obvious. Murray and Best have shown that if a venous segment is placed within an arterial defect fibrin does not form along the suture lines and the channel remains clear. In cases of embolectomy of the extremities it has been definitely shown that the results are infinitely superior when heparin is used postoperatively. There is less likelihood of a clot reforming in the vessel and obstructing the lumen. Best has shown in moving pictures that when blood is run through a glass chamber on which scratch marks occur, if heparin is not used thrombi will form on the roughened area and break off and be car-

ried into the blood stream. When heparin is used, however, while very small clots may form, they do not propagate, and emboli are not produced. As heparin has to be administered intravenously in physiologic solution of sodium chloride over a period of three to five days as a prophylactic measure to prevent thrombosis, its use becomes an economic burden. It costs about $8 a day, and the attention of a nurse is needed to be sure that the saline solution is running and that the needle does not become dislodged. Fortunately the peripheral phlebitis usually produced if a needle is left in a vein does not occur with administration of heparin. For prophylactic treatment, therefore, in otolaryngologic practice, it would seem to me that the immediate operative procedure, the restriction of much fats and proteins in the diet and the administration of sodium thiosulfate might serve as a useful adjunct. After phlebitis has gained headway it is doubtful that any therapeutic means will compare with proximal ligation of the affected vein. This probably acts in two ways: It prevents propagation of the thrombus and the resultant danger of infarction, and it severs the lymphatic chain which also carries the bacteria along.

Murray reported that pulmonary embolism, a common complication, occurring in 15 per cent of 300 cases of acute thrombophlebitis, did not occur in 81 cases in which heparin was used. In 5 cases of migrating thrombophlebitis the symptoms disappeared more slowly than in cases of the more acute forms of the disease. In 3 of these cases there were recurrences of the diseases within a few months.

It is uncertain why heparin should produce these effects on thrombophlebitis. The only effect that heparin could have is to prevent extension of thrombosis. It is possible, therefore, that healing of the lesion already present is allowed to take place and that with prevention of extension of the process the acute inflammation recedes and the symptoms disappear with it.

Murray and Best have shown that in 29 cases in which an initial embolus has formed, if heparin is promptly administered no further emboli are apt to occur. I quote from Murray:

> Up to the present time, 29 patients with massive pulmonary embolism have been treated with heparin. These patients all presented the usual picture of the disease, with sudden onset, marked collapse and rapid pulse. Several had multiple embolisms with infarcts which were demonstrated on clinical and roentgen examination. Heparin therapy was instituted at intervals of a few hours to several days after the attack. The results were surprisingly good. Of the 29 patients, none has died of embolism, although death seemed imminent for some. The dyspnoea and other signs of the disease improved rapidly, and within twenty-four hours all the patients stated that they were improved. From that time on the symptoms rapidly disappeared. With one possible, but unproved, exception, none of the patients had further emboli. With this disease, as with thrombophlebitis, it is impossible to predict the outcome, as it is well known that 80 per cent of patients recover.

In this group, however, the involvement in many of the cases was serious, and, judging from our experience with the disease, was of the type for which the prognosis is extremely bad. It is possible that the psychic effect of some active form of treatment was important, but this factor is difficult to evaluate.

Two other patients who were treated with heparin died, and postmortem examinations were made. The first died of general peritonitis following a partial gastrectomy, and thrombophlebitis and pulmonary embolism occurred as postoperative complications. At autopsy the infarcts of the lungs were seen to have been resolving, and there was no recently formed clot or embolus either in the pulmonary vessels or in the peripheral venous system. An area in the left common, external iliac and femoral veins, the region of which was obviously the site from which the embolus had come, showed some remaining thrombus, but this was firmly attached to the wall of the vein.

Recently interesting work has been reported by Kelson and White, of Boston, who have been treating malignant endocarditis due to Streptococcus viridans with sulfapyridine and heparin, with astonishing results. They have been able to obtain resolution of the process in a number of cases in which four blood cultures had yielded bacteria; the blood cultures were made sterile and the patient evidently recovered from the disease for several months. Their work is yet too recent to enable one to determine its true value. They have found that, whereas often for a week after administration of sulfapyridine the patient's temperature would return to normal, later there would be a recrudescence of the disease, and the patient would become sulfapyridine fast. They have also shown that heparin alone has been able to create the desired results. It would seem from their recommendations that sulfapyridine should not be administered until one is ready to utilize heparin. The combination of the two apparently is effective. Examination of the hearts of 2 patients who had died, apparently not of the original sepsis, showed that the vegetations on the valves had become smooth and thin, in contradistinction to the usual picture, in which the vegetations show considerable fibrin formation with relatively few bacteria between the fibrin fibrils. They concluded that the treatment had shown definite healing processes. It may well be that the combination of a sulfanilamide radical with heparin may serve as a real adjunct in the treatment of septic processes in the head and neck. It must be remembered, however, that, as heparin is a sulfur radical, a certain amount of care must be exercised in its use in combination with sulfanilamide or like medicaments.

In cases of very early thrombophlebitis of the extremities heparin alone has seemed at times to produce almost miraculous cures, evidenced by immediate subsidence of fever, diminution in swelling of the extremity and disappearance of the local tenderness. Murray and Best claimed that in cases of late thrombophlebitis (not, however, septic) they have had satisfactory results. My own experience in this, however, has been disappointing.

Although in most cases of peripheral embolism blockage is incomplete and in others there is sufficient collateral circulation, gangrene of the extremities often develops, necessitating amputation. Many patients respond to treatment with the pavaex machine, but in the experience of Murray, myself and others this device will not prevent gangrene in all cases. It is suggested, therefore, that if operation is done within the first twenty-four hours and combined with the use of heparin, better results may be expected. If the patient is seen after twenty-four hours, heparin should be utilized as well as the pavaex machine and may also be combined with papaverine to create capillary hyperemia.

Twelve arterial embolectomies have been done by Murray with heparin used postoperatively. In all cases this procedure was successful if the lumen was cleared completely and the circulation restored. Heparin was injected into the vessel at the site of operation, and the injection was continued intravenously for from three to fourteen days. In all 12 cases the vessels remained patent, and the circulation was maintained in the extremities.

This discussion would be incomplete if I did not mention some of the later theories in the creation of recurrent phlebitis and migratory phlebitis as seen in the extremities. Thompson, of New Haven, has presented the theory that phlebitis of the extremities in many cases is due to some form of dermatophytosis. He stated that this may be an allergic response. Irving Wright, of New York, stated the belief that dermatophytosis is a real factor in the production of this type of phlebitis and that it may be due either to a true fungous infection or to a virus. Certainly I have seen enough cases of migratory or peripheral phlebitis, not of the postoperative type, associated with dermatophytosis to make me believe that there is a real relation. Often cure of the dermatophytosis will cause subsidence of the recurring phlebitis. There may be also an endocrine relation which physicians are not as yet sufficiently conversant with to present as a definite fact.

SUMMARY

To sum up my own experience with heparin:

I have had indifferent results in the treatment of late phlebitis. I have had excellent results with embolectomy and with sudden vascular occlusion. At the City Hospital in a recent case of diabetic occlusion of the great toe, with a white distal phalanx suggesting impending gangrene, after administration of heparin the distal phalanx became red and warm, and there was a blister, with some sloughing of the tissues over the proximal phalanx. Amputation was unquestionably averted.

My associates and I are at present experimenting with heparin at the City Hospital in cases of thromboangiitis and diabetes with impending gangrene. A summary of the results of this work will probably be published later. So far the results have been encouraging.

I have not had experience in the use of heparin for pulmonary emboli, largely because when the clotting factors have been high I have used, prophylactically, restricted diet and sodium thiosulfate, and have been fortunate enough not to encounter pulmonary emboli.

For the septic thrombosis occurring in association with otolaryngologic conditions it would seem advisable to combine chemotherapy with heparin. Heparin itself has no antiseptic action and, as has been shown in cases of septic endocarditis, has little effect unless combined with sulfapyridine. The combination of the sulfanilamide group and heparin should aid in prevention of the spread of septic emboli and in stabilization of the clot when it is impossible to ligate the vein proximally.

USE OF SALT PORK IN CASES OF HEMORRHAGE

A. J. CONE, M.D.
ST. LOUIS

The problem of hemorrhage is ever important in treatment of the ear, nose and throat. As a general statement it may be said that the etiologic factors cover a wide range, varying with the age group concerned. Degenerative cardiovascular lesions with hypertension, the vascular changes associated with rheumatic fever, trauma, whether accidental or resulting from a surgical procedure, and blood dyscrasias in which there is some alteration or deficiency in some thromboplastic substance or substances all present possibilities which mean trouble when related to the field of the ear, nose and throat. Osler [1] stated: "In haemophilia the nose ranks first of the mucous membranes from which bleeding is common. It occurs in all forms of chronic anaemias. It precedes the onset of certain fevers and is associated in some special way with typhoid fever. Vicarious epistaxis has been described in suppression of the menses."

In cases of hemorrhage, fear has been recognized from time immemorial. Osler [2] stated: "Aretaeus remarks, 'the patient despairs from the first and needs to be strongly reassured.'" He further stated: "The truth, 'Das Blut ist ein ganz besonderer Saft,' is strikingly emphasized by the frightened state of the patient. Rest of body and peace of mind, 'quies, securitas, silentium' of Celsus should be secured." The foregoing quotations are made in reference to pulmonary hemorrhage. I have found them equally true in any case of hemorrhage.

There are many intangible things about hemorrhage which are not subject to scientific control or accurate evaluation. The experiments related to hypnosis and suggestion should not be overlooked.

The conclusions of Schindler, as quoted by Dunbar [3] in his book "Emotions and Bodily Changes," are:

The development of spontaneous hemorrhages by way of purely nervous influences is established. In hysteria especially, small ecchymoses are of frequent occurrence

From the Department of Otolaryngology, the Washington University School of Medicine.

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1. Osler, W., and McCrae, T.: The Principles and Practice of Medicine, ed. 9, New York, D. Appleton and Company, 1920, p. 604.

2. Osler and McCrae,[1] p. 632.

3. Dunbar, H. F.: Emotions and Bodily Changes, New York, Columbia University Press, 1935, p. 385.

and not infrequently large and constantly recurring extravasations occur which apparently may be the expression of certain psychic processes. The resulting disease picture is extremely serious. Since it is curable, its diagnosis is important. Knowledge of these phenomena is of the greatest forensic interest. Therapy even of the serious cases is promising. Of course, only psychotherapy comes into consideration. Peculiarly enough, all cases that were approached psychotherapeutically, seven in all, were cured by simple suggestion or hypnosis. It is highly probable that there occur also not infrequently internal hemorrhages (lungs, intestines, kidneys, etc.) of milder or more dangerous form that are psychically conditioned. A classification of hemorrhagic diatheses must be constructed anew on the basis of this knowledge.

I have rarely been able to allay fear when hemorrhage from the upper part of the respiratory tract has required introduction of pressure packs or coagulation by electrical or chemical means. In many instances, especially with excitable patients or critically ill children, the shock attendant on forceful restraint and treatment introduces an element not readily forgotten by the patient or the physician. In the case of pressure packs there is commonly a recurrence of bleeding at the time of removal of the gauze. Reintroduction of such packs, frequently necessary in the case of a patient with a blood dyscrasia, such as leukemia, becomes a matter of major importance in management of the patient and adds despair to an already hopeless condition.

Other dangers, such as the occurrence of infection in the sinuses or in the ears, are ever present. Hilding [4] has reported a case of fatal meningitis following the use of nasal packs for control of epistaxis. I have seen a patient in whom the control of a nasal hemorrhage was followed by acute otitis media, mastoiditis, thrombosis of the lateral sinus and sepsis, with weeks of hospitalization and repeated operations required to combat the numerous complications of the sepsis.

Since the publication in 1927 of a paper by Hurd [5] on the use of salt pork to control hemorrhage, I have used his method in many cases of nasal hemorrhage. The ease of use, the uniformity of good results and the absence of any complication have made it an invaluable procedure.

In my personal experience severe recurrent nasal hemorrhage has been seen most frequently in children with rheumatic fever. The incidence of nasal hemorrhage in rheumatic persons has been reported by Gibson,[6] who stated that "epistaxis is frequent in active rheumatic heart

4. Hilding, A.: False Response to the Jugular Compression (Tobey-Ayer) Test Due to Anomaly of the Lateral Sinus, Arch. Otolaryng. **26**:143-145 (Aug.) 1937.

5. Hurd, L. M.: The Use of Salt Pork to Control Hemorrhage, Arch. Otolaryng. **6**:447-448 (Nov.) 1927.

6. Gibson, S., in Brennemann, J.: Practice of Pediatrics, Hagerstown, Md., W. F. Prior Company, Inc., 1936, vol. 3, chap. 13, p. 43.

disease . . . it should be considered in any case in which repeated and profuse nose bleeds have occurred."

Swift [7] stated: "In certain years we have observed it with great frequency with, preceding and accompanying acute attacks." Levine, Andren and Homans [8] reported epistaxis as three to four times as frequent in rheumatic patients as in a nonrheumatic control group. They stated: "Epistaxis may occur for years preceding the first definite attack of rheumatism."

Kaiser [9] listed epistaxis as occurring in 15 per cent of a series of 1,240 cases of rheumatic fever.

McCulloch [10] has repeatedly stressed the importance of epistaxis in the rheumatic child.

I have reviewed a group of 187 children, most of whom I have had the privilege of following in the St. Louis Children's Hospital service. In this group epistaxis occurred in 44 cases. The epistaxis had been a recurrent complaint of most of these children. It was rarely associated with chorea but occurred usually in the proliferative stages of the disease.

The changes in the peripheral vessels of the rheumatic patients have been described by Von Glahn and Pappenheimer,[11] who stated that the lesions are "characterized by exudation of fibrin into and about the vessel, by destructive cellular reaction in the adjacent tissue and by the absence of thrombosis."

I have removed a portion of the septal membrane of a rheumatic child and found marked changes in the vessels in serial sections, which I feel were of sufficient degree to explain the basis of the severe hemorrhage.

Swift [12] stated: "It thus appears evident that involvement of the vascular tree may eventually prove almost as important as that of the myocardium and valves." Rhinologists, then, have an important clinical group of patients the recognition of whose condition is essential and whose management is limited by the very nature of the disease. These patients are in an age group in which cooperation and confidence are easily lost.

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7. Swift, H. F.: Rheumatic Fever, in Nelson's Loose Leaf Living Medicine, New York, Thomas Nelson & Sons, 1927, vol. 1, p. 429E.

8. Levine, S. A.; Andren, T., and Homans, K. A.: Nosebleed and Vomiting in Rheumatic Individuals, New England J. Med. **203**:832-834 (Oct. 23) 1930.

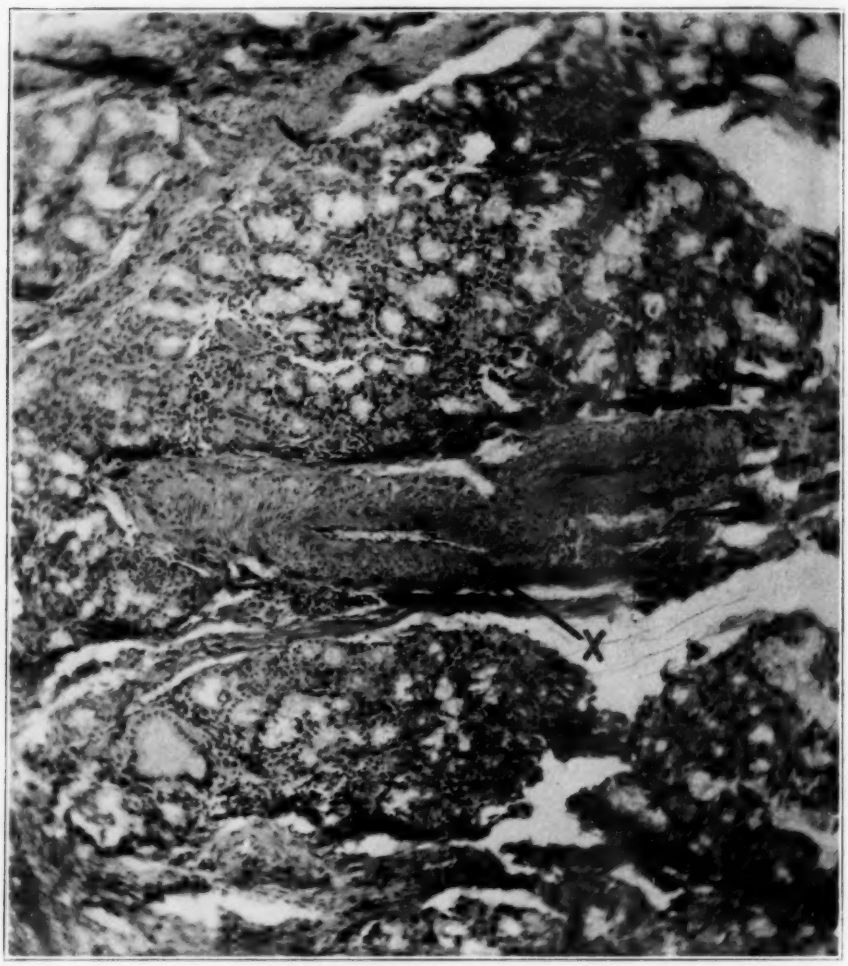
9. Kaiser, A. D.: Factors That Influence Rheumatic Disease in Children, J. A. M. A. **103**:886-892 (Sept. 22) 1934.

10. McCulloch, H.: Personal communication to the author.

11. Von Glahn, W. C., and Pappenheimer, A. M.: Specific Lesions of Peripheral Blood Vessels in Rheumatics, Am. J. Path. **2**:235-249 (May) 1926.

12. Swift,[7] p. 416.

In using wedges of salt pork in the treatment of children it has not been uncommon in the St. Louis Children's Hospital service to have a child request that salt pork be inserted in his nose with the first sign of a nosebleed. At a later time of election, when the threat of



Photomicrograph of a section of the nasal membrane removed from a rheumatic patient whose condition was inactive. The vessel wall shows thickening. The subendothelial region shows hyaline change (*X*). There is no evidence of thrombosis in the vessel.

severe hemorrhage has passed, more permanent procedures, such as submucous elevation of a septal flap, may be carried out with full cooperation of the child.

Wedges of salt pork have saved a great deal of time and energy when used in controlling nasal hemorrhage, as seen in cases of leukemia, hemophilia and other blood dyscrasias. Use of such wedges has been equally efficient in controlling the bleeding associated with hypertension. I have used them in controlling violent hemorrhage occurring with the onset of measles or typhoid fever and during the third stage of labor; the relief of anxiety of the patient and all concerned was particularly gratifying.

In many instances salt pork promptly checked bleeding after other methods had failed. This was true in 1 case in which violent nasal hemorrhage occurred five days after submucous resection and partial removal of a middle turbinate.

J. T., a 23 year old white woman, entered the Barnes Hospital May 20, 1931, for a submucous resection of the nasal septum and partial removal of the right middle turbinate. There was no history of hemorrhage or irregular menstruation. The time of operation supposedly was two weeks after normal menstruation. The postoperative course in the hospital was uneventful, and the patient was discharged on the second postoperative day. On the fifth postoperative day she was brought back to the hospital because of severe hemorrhage, which had required gauze packing by another physician before he was able to transport her to the hospital. She stated that she had started to menstruate on the evening before and that nasal bleeding came on shortly after the onset of the unexpected menses. The patient was given two doses (totaling 5 cc.) of hemostatic serum intramuscularly. Her blood pressure fell to 60 systolic and 40 diastolic. As a supportive measure 500 cc. of 10 per cent dextrose and acacia was given intravenously. Morphine was administered hypodermically. Bleeding continued. A lamb's wool pack was inserted postnasally; gauze packs were inserted anteriorly against the postnasal pack. The blood pressure rose to 137 systolic and 80 diastolic. The red blood cell count had fallen from 5,100,000 to 2,500,000 per cubic millimeter. It was estimated that she had lost at least 600 cc. of blood in the twelve hours between noon and midnight of the first day after return to the hospital. During the next day (second day after readmission) the patient had a little bleeding and vomited several times, old blood being a large part of the vomitus. She was given 1,500 cc. of saline solution subcutaneously. Morphine was used to allay apprehension. Then 500 cc. of whole blood was given by the multiple syringe cannula method, it being felt that whole blood might be of greater benefit than citrated blood. At 3:30 p. m. of the second day the nasal bleeding recurred, in spite of the postnasal and nasal packs. The temperature went up to 39 C. At 4 p. m. of the second day the nasal packs were removed, and a salt pork wedge was placed in the right side of the nose. From this time on there was never more than a little serohemorrhagic drainage. On the third day after readmission the postnasal pack was removed. On the fourth day the salt pork was removed. There was no further bleeding. Recovery was uneventful.

Hurd [5] stated: "It is hard to say just what the action of the pork is, since several factors are present; namely, pressure, salt, tissue juice, and fat."

In an attempt to determine the mode of action I have made extracts of salt pork, fresh pork and fresh pork muscle and checked the effect

on the clotting time. Salt pork was used in dilutions of 1:2, 1:5, 1:10 and 1:20. The presence of thromboplastic substance was indicated, since all blood specimens clotted in close to three minutes. The normal time of coagulation of untreated blood was from seven to ten minutes. Because there was less than a fifteen second variation in the effect of different dilutions, it was considered that the high salt concentrations decreased the clotting time in the salt pork test. Therefore, the same procedure was repeated with fresh pork fat and fresh pork muscle extracted in physiologic solution of sodium chloride. Rabbit's lung, extracted in the same manner as the pork preparations, produced clotting in forty seconds. The following results were noted:

	Fresh Pork Fat	Fresh Pork Muscle	Rabbit's Lung	Untreated Blood	Physiologic Solution of Sodium Chloride (Control)
Undiluted.....	1 min. 30 sec. 1 min. 15 sec.	55 sec. 40 sec.	40 sec.	7 min. 8 min.	8 min.
Dilution 1:4 with physiologic solution of sodium chloride	2 min. 5 sec. 3 min. 30 sec.	62 sec. 2 min. 45 sec.	.....	10 min.	

It seems reasonable to assume, then, that pork contains some thromboplastic substance which is not enhanced by the brine preparation; that pork muscle is a better source than pork fat as far as thromboplastic substance is concerned, and that neither is as good a source as rabbit lung. However, there are other factors which must be considered. Salt pork is easily kept in brine and does not disintegrate. Pork fat does not harbor the parasites that might be present in lung tissue or in muscle. It is an accepted fact that salts in high concentration prevent bacterial growth. Hurd [5] reported that culture of a random specimen of salt pork kept in brine for six months showed only a few colonies of hay bacillus. I have made repeated cultures of salt pork of different ages and have not found a pathogenic organism.

Hurd's summary,[5] as reported in 1927, presents the peculiar advantages of the salt pork preparation: "Larding pork is cheap, common, and easily obtained. It keeps well and is handy to use. It is superior to the usual nasal pack in that it is easier to introduce, and it is more comfortable for the patient whose only complaint is that the salt causes smarting for a few minutes. Pork promptly stops the bleeding, and seems to have the property of preventing recurrence."

Case Reports

BACILLUS PROTEUS IN OTITIS MEDIA, COMPLICATED BY MASTOIDITIS, MENINGITIS AND BACTEREMIA

ERWIN NETER, M.D., AND ROBERT A. CHAIT, M.D., BUFFALO

Members of the genus Proteus are widely distributed in nature. They may also be present as saprophytes in the intestinal tract of normal human beings. Only rarely are they encountered as the primary causative agents in diseases of man. More frequently they play the role of secondary invaders. Thus they may be found in association with infections of the urinary tract, perforative peritonitis, cholecystitis, infections of the ear and mastoid, conjunctivitis, puerperal fever, traumatic infections and other anomalies. Although the members of the genus Proteus have been frequently isolated in cultures of material from patients with enteric diseases, their significance in the pathogenesis of these conditions, particularly summer diarrhea of children and food poisoning, is not fully understood.

It is interesting to note that only rarely do members of the Proteus group invade the blood stream. The bacteremia may originate from any lesion and complicate a variety of diseases. MacKenzie and Hawthorne [1] (1933), who reported on bacteremia due to Bacillus proteus complicating infections of the urinary system, collected 17 cases from the literature and added 1 observed by themselves. Occasionally invasion of the blood stream by B. proteus may follow operations, e. g. prostatectomy (Wallace and Dudgeon; [2] Buckley [3]). Only a few cases of bacteremia due to B. proteus and originating from infection of the ear or peritonsillar abscess have been described in the recent literature (Kernan; [4] Pagniez, Plichet and Décourt; [5] Smith and Miller; [6] McGovern; [7] Vallée and Giroux,[8] and Gerzog [9]).

Purulent meningitis due to B. proteus, either as the only pathogenic micro-organism or together with others, is as rare as bacteremia caused

From the Bacteriological Laboratories and the Department of Otolaryngology of the Children's Hospital and of the University of Buffalo School of Medicine.

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2. Wallace, C. S., and Dudgeon, L. S.: On Bacillus Proteus Infection, More Especially in Relation to a Case of Proteus Septicaemia Associated with Femoral Thrombosis, Pulmonary Embolism and Recovery, Lancet **1**:597-599, 1915.

3. Buckley, T. I.: Bacillus Proteus Septicemia, California & West. Med. **38**:436-437, 1933.

4. Kernan, J. D.: Two Cases of Infection by Bacillus Proteus, Laryngoscope **32**:304-310, 1922.

5. Pagniez, P.; Plichet, A., and Décourt, P.: A propos d'un cas de septicémie à "proteus," Bull. et mém. Soc. méd. d. hôp. de Paris **56**:270-274, 1932.

6. Smith, A. B. P., and Miller, S.: Lateral Sinus Thrombosis Due to Bacillus Proteus, Lancet **1**:862, 1935.

(Footnotes continued on next page)

by that organism. In 1927 Neal and Abramson [10] reviewed the literature on this subject; they collected 7 cases and added 2 of their own. Since their report a few additional cases have been recorded, by Bewley and Horgan,[11] Taylor,[12] Herzig,[13] Manicatide and Rosenkrantz,[14] Kortenhaus,[15] Cathala and Gabriel,[16] Pangalos and Doucas [17] and Calhoun,[18] and finally by Peluffo, Ledesma and Deagustini.[19] Furthermore, a case of meningomyelitis caused by B. proteus vulgaris has been reported by Lévy-Valensi, Sèze and Imbona.[20] It may be briefly mentioned in this connection that B. proteus has also been found in the cerebrospinal fluid of patients with typhus fever (Anderson [21] and Rosca [22]). Finally, the organism has been encountered as the causative agent in brain abscess, complicating aural infection (Gallagher [23]).

In view of the rare occurrence of invasion of the blood stream and of the meninges by members of the genus Proteus, we should like to present a case of otitis media complicated by mastoiditis, meningitis and septicemia, in which B. proteus vulgaris was isolated in cultures of material from all lesions.

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8. Vallée, A., and Giroux, M.: Mastoïdite aiguë à Protéus vulgaris: Septicémie, pleurésie, Laval méd. **2**:107-112, 1937.

9. Gerzog, B. G.: Bacillus Proteus in Otogenic Infections, Secondary Mastoiditis, Thrombosis of the Lateral Sinus and Bacteremia, Arch. Otolaryng. **30**: 275-279 (Aug.) 1939.

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11. Bewley, G., and Horgan, E. S.: A Case of Meningitis Associated with B. Proteus, Lancet **1**:819-820, 1927.

12. Taylor, J. F.: Bacillus Proteus Infections, J. Path. & Bact. **31**:897-915, 1928.

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20. Lévy-Valensi, J.; Sèze, S., and Imbona: Méningo-myélite aiguë à "Proteus vulgaris," Bull. et mém. Soc. méd. d. hôp. de Paris **54**:677-680, 1938.

21. Anderson, J. M.: Bacillus Proteus in Cerebro-Spinal Fluid of Typhus Fever, J. Path. & Bact. **24**:478-479, 1921.

22. Rosca, V.: Contribution à l'étude de la structure cytologique des bactéries, Arch. roumaines de path. expér. et de microbiol. **10**:267-279, 1937.

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REPORT OF A CASE [24]

A girl aged 14 years was admitted to the Children's Hospital on Nov. 22, 1938, presenting a history of intermittent otitis media on the right side for two years. Four days before her admission she complained of pain in the right ear and of right frontal headache. She vomited repeatedly.

Physical Examination.—On admission the patient had a temperature of 104.2 F. A small area of redness was observed over the right mastoid tip, but there was no tenderness. Purulent material was draining from the right ear. The drum was perforated. The left ear was free of discharge. There was a suggestion of weakness of the facial muscles on the right side. Neither strabismus nor nystagmus was observed. The external ocular movements were normal. The neck was rigid. A positive Kernig sign was elfcited on both sides. There was no Babinski reflex. Results of the remainder of the physical examination were essentially normal.

Laboratory Examination.—A blood count revealed: a hemoglobin value of 95 per cent and a leukocyte count of 14,000, with polymorphonuclears 85 per cent (marked shift to the left) and lymphocytes 15 per cent. The urine was acid and showed a trace of albumin but no sugar. Microscopic examination showed the leukocytes to be rare. A tuberculin test gave a positive reading. The reaction to the Kahn test was negative. Roentgenograms showed sclerosis of the right mastoid process and to a lesser degree of the left.

A spinal tap was performed. The cerebrospinal fluid was under increased pressure. It was cloudy, containing 3,860 leukocytes. The reaction to the Pandy test was markedly positive. Copper reduction was prompt. Smears of the sediment of the spinal fluid showed no micro-organisms. Cultures for aerobic as well as for anaerobic organisms were sterile.

A culture of blood taken on the day of admission, before the operation, yielded B. proteus vulgaris (two colonies per cubic centimeter of blood). (The characters of this strain are given later in this report.)

Operation and Course.—On the day of admission, a mastoidectomy on the right side was performed (Dr. D'Arcy McGregor). Thick, creamy yellow pus welled up into the wound when the cortex was uncapped. The antrum was filled with thick, spongy granulation tissue. Cholesteatomatous material was found on the floor of the aditus. Several necrotic areas were present in the plate covering the lateral sinus. A perisinal abscess was uncovered. The wall of the sinus was thickened. The dura mater of the middle fossa was exposed over an area as large as a 50 cent piece; part of the floor and lateral wall of the middle cranial fossa was removed. The dura, which was thickened and dirty greenish, was incised. The wound was packed with iodoform gauze. Cultures were made of material taken from the right ear and from the mastoid cavity. (The findings are given later.)

The day after the operation the patient seemed clinically improved. Her temperature had dropped to 100 F. On November 24, however, it rose to 104 F. and remained elevated (ranging from 101 to 106 F.) for the remaining days of her illness.

On November 25 a second spinal tap was performed. On this occasion the spinal fluid was cloudy and contained a large amount of fibrin. The sugar content of the spinal fluid was now reduced to 12 mg. per hundred cubic centimeters. The sediment showed numerous gram-negative bacilli, which were identified as

24. From the service of Dr. J. Fairbairn.

B. proteus vulgaris. A second blood culture revealed no growth. A culture of the urine made on November 25 failed to show B. proteus.

In view of the fact that the patient had purulent bacterial meningitis and bacteremia, neoprontosil (prontosil soluble, the disodium salt of 4-sulfamidophenyl-2′-azo-7′-acetylamino-1-hydroxynaphthalene-3; 6′-disulfonic acid) was given intramuscularly and sulfanilamide (0.8 per cent in physiologic solution of sodium chloride) intravenously. In spite of this treatment, the patient died on the eighth day following admission to this hospital.

Necropsy.—The postmortem examination revealed the presence of acute diffuse purulent leptomeningitis, subdural empyema of the right half of the posterior cranial fossa, multiple hemorrhagic areas in both lungs and a septic condition of the spleen. Thrombophlebitis of the dural sinuses was not present.

Bacteriologic Findings.—Culture of the material from the ear and from the mastoid revealed streptococci that were overgrown by a gram-negative bacillus. Attempts to isolate and identify the streptococcus failed. The bacillus was identified as B. proteus vulgaris. An identical gram-negative bacillus was obtained from the culture of the blood (Nov. 25, 1938) and from that of the spinal fluid (obtained on Nov. 25, 1938). The bacillus was motile and showed characteristic spreading on agar plates. It grew well on the ordinary culture mediums, including Endo agar. The organism peptonized litmus milk. It produced acid and gas from dextrose, maltose and sucrose but not from lactose and mannitol. It did not form indol after twenty-four hours' incubation at 37 C.

COMMENT

This case of B. proteus infection of the middle ear and mastoid, complicated by purulent meningitis and septicemia, is a rare instance that conclusively illustrates the potential pathogenicity of this micro-organism. Although there can be little doubt that B. proteus was not the only micro-organism responsible for the disease, the demonstration of its presence in all the lesions involved indicates that it was an important contributing factor. The primary causative agent of the otitis media probably was the streptococcus seen in the material obtained from the middle ear and mastoid and in the purulent exudate covering the brain.

The attempt to cure the generalized infection by means of chemotherapeutic substances of the sulfanilamide group was not successful. It is interesting to note, however, that the second blood culture made after this treatment was begun no longer revealed B. proteus. Clinically, sulfanilamide is effective in the treatment of infections of the urinary system due to B. proteus. However, it is necessary to keep in mind that the concentration of sulfanilamide in the urine usually is much higher than that obtained in the blood or in various tissues and tissue fluids. In the case here reported the concentration of sulfanilamide in the blood ranged only between 1 and 2 mg. per hundred cubic centimeters.

SUMMARY

A case of otitis media complicated by mastoiditis, meningitis and septicemia is presented. B. proteus vulgaris was isolated in cultures of material from all lesions involved. The pathogenicity for man of the members of the genus Proteus, particularly in regard to meningitis and septicemia, is discussed.

VEGETABLE FOREIGN BODY IN THE LUNG FOR A MONTH

Successful Removal; Recovery Complicated by Measles

CLYDE E. HARNER, M.D., LONG BEACH, CALIF.

It is rare for vegetable material, such as peanuts, corn or beans, to remain in the lung of a young child for long without causing fatal bronchopneumonia.

I am reporting a case in which such a foreign body remained in the right main bronchus for a month and was successfully removed.

REPORT OF A CASE

K. W., a baby aged 21 months, was given a corncob to play with; he suddenly choked and had a paroyxsm of coughing, becoming cyanotic for a while. The mother examined the corncob, to which a few kernels of corn were still attached, and saw that apparently a small piece of it was missing. She tried various measures to dislodge the substance, even to holding the child by the feet head downward, but was unsuccessful.

The child continued to have paroxysms of coughing, especially on lying down. Fever developed, and the baby was irritable and fretful. The family physician said it was improbable that any of the cob had been aspirated, and, since the family lived on a ranch in a somewhat isolated community, nothing further was done until one month later.

The child had lost weight, had fever daily, the temperature being several degrees above normal, and continued to have paroxysms of coughing during which he was cyanotic.

He was referred to me. A roentgenogram showed emphysema of the right lung, slight displacement of the heart and mediastinum to the left, depression of the diaphragm on the right side and increased density of the left side of the chest. A diagnosis was made of a nonopaque foreign body blocking the right main bronchus.

The child was given $\frac{1}{16}$ grain (3.75 mg.) of morphine and $\frac{1}{800}$ grain (0.075 mg.) of atropine one hour before the bronchoscope was used. Without anesthesia of any kind and with a 5 mm. bronchoscope, the right main bronchus was found to be closed by exuberant granulation tissue. This having been removed and a hook being used, a small gray mass was uncovered, which proved to be a piece of corncob with a small macerated kernel of corn attached. No other fragments were found.

The patient immediately began to improve, and chest sounds became more normal until about ten days had elapsed, when roughened respiratory sounds began to recur and the temperature rose to 104 F. It was naturally suspected that there was a small piece of foreign material remaining, but the rash of measles appeared. After recovery from this condition, it was apparent that the respiratory tract was normal and that all of the foreign body had been removed.

517 Professional Building.

Clinical Notes; New Instruments and Technics

IRRIGATION OF THE SPHENOID SINUS

G. Edward Tremble, M.D., Montreal, Canada

The sphenoid sinus was not recognized as a possible source of infection until 1905.[1] At that time and later (in 1908 [2]), Onodi first described the anatomic connections between the posterior group of sinuses and the optic canal. As a result of his observations he felt that he had definitely established an etiologic relation between disturbances of vision and disease of the posterior ethmoid cells and the sphenoid sinuses.

Loeb [3] in 1909 corroborated Onodi's work, and Schaeffer [4] in 1920 again emphasized this as a definite fact. They all showed that it is not uncommon for the posterior ethmoid cells and the sphenoid sinus to encroach on the optic canal, making it "abnormally small," a condition which may lead to serious pressure on the nerve, with ophthalmologic symptoms.

In 1927, Crane [5] expressed the opinion that not only is there a likelihood of direct extension of infection from this relation but that simple inflammation of the sinuses is sufficient to cause disturbances in the nerve. Dehiscences of the bone, particularly in the roof, have been cited as the method of extension. In 1928, just before his death, White [6] expressed the belief that invasion of the optic nerve is always hematogenous.

There has been considerable controversy as to whether retrobulbar neuritis is caused by a sinal infection or by multiple sclerosis. In 1934, Lillie,[7] of the Mayo Clinic, questioned the importance of the sinuses in relation to disturbances of

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Dr. S. Hanford McKee supplied many of the references used in this article.

Read before the Otolaryngological Section of the Medico-Chirurgical Society of Montreal at the Royal Victoria Hospital, Feb. 15, 1940.

1. Onodi, A.: Die Nebenhöhlen der Nase, Vienna, Alfred Hölder, 1905.

2. Onodi, A.: The Optic Nerve and the Accessory Cavities of the Nose, Ann. Otol., Rhin. & Laryng. **17**:1 (March) 1908.

3. Loeb, H. W.: The Cubical Capacity and Superficial Area of the Sphenoidal Sinus, Ann. Otol., Rhin. & Laryng. **21**:1 (Jan.) 1912; A Study of the Anatomic Relations of the Optic Nerve to the Accessory Cavities of the Nose, ibid. **18**:243 (June) 1909.

4. Schaeffer, J. P.: The Nose, Paranasal Sinuses, Naso-Lacrimal Passageways, and Olfactory Organ in Man, Philadelphia, P. Blakiston's Son & Co., 1920, chap. 5, p. 175.

5. Crane, C. G.: Anatomic, Pathologic and Clinical Relationship of Posterior Sinuses to Optic Neuritis, Ann. Otol., Rhin. & Laryng. **36**:201-240 (March) 1927.

6. White, L. E.: Location of the Focus in Optic Nerve Disturbances from Infection, Ann. Otol., Rhin. & Laryng. **37**:128-164 (March) 1928.

7. Lillie, W. I.: Clinical Significance of Retrobulbar and Optic Neuritis, Am. J. Ophth. **17**:110-119 (Feb.) 1934.

the optic nerve and stressed multiple sclerosis as the commonest cause in his series of cases. Others have made similar reports. At present there seems to be considerable confusion as to the true cause of retrobulbar neuritis.

Whether or not it is due to infection in the sphenoid sinus or to multiple sclerosis, the fact remains that in many cases there is a dramatic and rapid improvement after the sphenoid sinus is opened. The results after operation are often spectacular even when no abnormality is found except perhaps a thickening of the mucous membrane of the sphenoid sinus.

Investigation of the sphenoid sinus through the natural ostium is often difficult and at times impossible. The anterior wall of the sphenoid sinus slopes downward and backward and consists of two portions, the nasal and the ethmoid. The nasal portion is about one third or more of the thin anterior wall close to the septum, which presents in the olfactory fissure and in the sphenoethmoidal recess. The position of the natural ostium is not constant, but it is always situated in the nasal portion of the anterior wall, usually in the upper third and close to the septum. Occasionally the ostiums on the two sides are not on the same level, and the distance from the floor of the sinus is also variable. However, in the vast majority of cases the opening is but a few millimeters below the roof of the nasal cavity. Only in exceptional cases can this be seen through the narrow slit available between the middle turbinate and the septum, so that it is necessary to depend on measurements and the sense of touch to find the aperture. Any enlargement of the middle turbinate or deflection of the septum increases the difficulty.

There are two methods of irrigating the sphenoid sinus: (1) through the natural ostium and (2) through an artificial opening in the anterior wall of the sinus. Various instruments have been used for this purpose, some curved and blunt, others straight and sharp. Jacob, cited by Laurens,[8] and Sieur[9] recommended a no. 1 eustachian catheter and a search for the natural ostium, while Watson-Williams[10] advocated a straight blunt cannula for puncture of the anterior wall, which in his hands proved successful.

The method of puncturing the thin anterior wall has certain advantages over the route through the natural ostium. For instance, with a cannula in place through an ostium which is very small and occluded by swollen mucous membrane it is impossible to apply suction, for, as Watson-Williams[11] mentioned, there is no "bunghole." Making an opening into the sphenoid sinus is analogous to irrigating the maxillary antrum through the lateral nasal wall under the inferior turbinate. Once the parts are anesthetized, it requires only one or two minutes to insert the cannula into the sphenoid sinus, and material aspirated in this way is less liable to be contaminated.

The instrument suggested has been found useful in outlining the sinus cavity, in obtaining culture material, for irrigation and treatment and as a guide in removing the anterior wall.

It consists of a cannula graduated in centimeters and slightly curved downward at the end. A double mark at 7 cm. indicates the distance from the anterior nasal spine to the anterior wall of the sphenoid sinus in the average adult. With

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8. Laurens, G.: Chirurgie, oto-rhino-laryngologique, Paris, G. Steinheil, 1906, p. 578.

9. Sieur, cited by Watson-Williams,[10] p. 175.

10. Watson-Williams, P.: Chronic Nasal Sinusitis, London, J. Wright & Sons, Ltd., 1933, p. 176.

11. Watson-Williams,[10] p. 175.

few exceptions, this distance, noted by Zuckerkandl and later by Hajek,[12] is a fairly constant measurement. A single line at 8 cm. marks the upper limit of the scale.

The caliber of the lumen is sufficient to allow for the extraction of thick pus, which is impossible with the smaller, malleable cannulas. The curve at the distal end of the cannula is such that it will penetrate the thin anterior wall at the safest spot, which is 10 to 12 mm. below the level of the roof of the sphenoid sinus.

The downward curve also insures entry into the lower half, or deeper part, of the sinus. (The difference between the upper, shallow portion of the sinus and the lower, deeper and much safer part has been stressed by Watson-Williams.[13])

A vertical tongue 11 mm. from the tip acts as a guard to prevent the instrument from penetrating too deeply into the sinus. This tends to avoid injuring the thin posterior wall which separates the sphenoid sinus from the pituitary fossa. Situated on the lower border of the cannula, this guard does not interfere in any way with vision.

The proximal end for attaching to the syringe or for suction is also turned downward slightly in order that an unobstructed view may be obtained along the

Fig. 1.—Cannula recommended.

barrel of the cannula. The handle is placed at a convenient angle, so that when the tip of the instrument is in the sphenoid sinus the handle is perpendicular if the patient's head is erect or horizontal if he is lying down.

The method of inserting the cannula is simple. According to Zuckerkandl, a line drawn from the anterior nasal spine diagonally upward across the lowest point or center of the middle turbinate touches the anterior wall of the sphenoid sinus just below the natural ostium. With this in mind and with the anterior end of the middle turbinate as the chief landmark, the instrument is introduced into the nose between the middle turbinate and the septum. The middle meatus is also an important guide, for if the cannula crosses the middle turbinate about 10 mm. from the top of this slit it should reach the most suitable place for puncture—the junction of the middle and the lower third of the anterior wall.

Many writers have mentioned the importance of entering the sphenoid sinus as close to the septum as possible to avoid injuring the lateral wall with its adjacent

12. Hajek, M.: Pathology and Treatment of the Inflammatory Diseases of the Nasal Accessory Sinuses, translated by J. D. Heitger and F. Hansel, St. Louis, C. V. Mosby Company, 1926, vol. 1, p. 83.

13. Watson-Williams,[10] p. 179.

cavernous sinus and optic nerve. However, in some instances that part of the septum which unites with the rostrum of the sphenoid is thick, and it is necessary to turn the handle of the instrument slightly outward, so that the tip is 2 or 3 mm. away from the septum, before perforating the wall. This also tends to avoid the septal branch of the sphenopalatine artery, which occasionally is a vessel of considerable size. When the cannula impinges against the anterior wall, it requires little pressure to enter the sinus. The sensation of slipping into a cavity is characteristic and is similar to going through the dura in doing a lumbar puncture.

Those who advocate a straight instrument recommend raising the proximal end as much as the nose permits once the point is "anchored" against the anterior wall of the sinus.

With the cannula suggested, this is necessary only to a slight degree, owing to the downward curve.

At times, deflections or spurs on the septum or polypi obstruct the view, but even in these cases, as long as the middle turbinate and the middle meatus are visible after cocainization, it is usually a simple procedure to reach the sphenoid sinus. Any marked obstruction which hides the view of the middle turbinate should, of course, be corrected first.

Occasionally a long-bladed speculum is needed to obtain a better view, but it is rarely necessary to remove any part of the middle turbinate.

The aforementioned landmarks are sufficient in the vast majority of cases, but once in a while it is important, even for an experienced operator, to know the level of the cribriform plate.

Dr. C. P. Martin, professor of anatomy at McGill University, made the following suggestions concerning the level of the cribriform plate. "The cribriform plate of the ethmoid is not completely flat but usually curves slightly upwards, both anteriorly and at the sides. The central and lowest part of the plate is virtually parallel with Reid's base line,[14] that is, a line cutting the lower margins of the bony orbits in front and passing through the middle of the external auditory meati behind. The cribriform plate is situated at the level of a line joining the inner canthi of the eyes."

In other words, from a practical point of view it is important to remember an imaginary line from the middle of the external canal opening to the lower margin of the orbit. Also, the level of the inner canthus of each eye is at least ½ inch (1.2 cm.) above Reid's base line in the average adult.

With this in mind, a parallel line ½ inch (1.2 cm.) higher therefore shows the approximate position of the cribriform plate. A simple method is to use two strips of adhesive tape exactly ½ inch (1.2 cm.) wide and about 2 inches (5 cm.) long. If the adhesive tape is placed on each side of the face, extending from in front of the ear to the edge of the orbit, so that the lower border of the tape lies along Reid's base line, the upper edge is well below the level of the cribriform plate. A black line along the upper edge of the tape is helpful at first, but after a time this is unnecessary.

After the remarks of Professor Martin, I consulted an optician regarding a spectacle frame which would show the level of the cribriform plate from the front in practically all cases.

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14. Reid's base line (Robert William Reid, Scotch anatomist, 1851) is a line drawn from the lower margin of the orbit to the auricular point (center of the aperture of the external auditory canal) and extending backward to the center of the occipital bone (Stedman, T. L.: A Practical Medical Dictionary, ed. 14, Baltimore, Williams & Wilkins Company, 1939).

Perhaps an exception occurs when a low-placed cribriform plate dips down into the nasal cavity, but this is a very rare occurrence. This alone should not be a serious danger, as a routine roentgen examination shows the situation clearly in the lateral view.

In such cases there is still a margin of safety of nearly a ¼ inch (0.6 cm.) once the anterior end of the middle turbinate bone is passed.

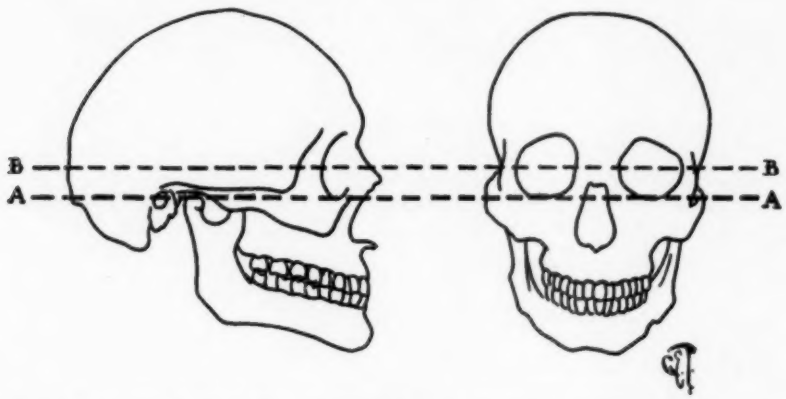

Fig. 2.—*A*, Reid's base line. *B*, level of the cribriform plate. (This line passes through the inner canthus of each eye.)

Fig. 3.—Spectacle frame recommended for ascertaining the level of the cribriform plate.

The most useful spectacle frame was found to be a light one, with plain glass and of average size. After many tests, the type shown in figure 3 seemed to fill all requirements.

In describing the frame, a few points should be mentioned. A horizontal black line is made across each glass, exactly on a level with the inner canthus of each eye. The ideal is one as shown, having the upper border of the arms, the black lines and the two elevations that rest on the sides of the nose in one straight line. It is true that the one frame does not fit perfectly in every case, but this can usually

be adjusted by slight pressure from above or by elevating the rims with a piece of adhesive tape over the bridge of the nose. The frame alone, without the tape, is often all that is necessary.

In using the spectacle frame it is important to have the patient sitting upright, with the head erect and the eyes straight ahead. In this position, after a little practice, it is a simple matter to insert the cannula blindly after passing the anterior end of the middle turbinate.

Apart from its usefulness in the aforementioned connection, the frame can be used in operations on the septum and the ethmoid cells. It is frequently helpful to know the level of the cribriform plate.

During the past year this instrument has been used successfully at the Royal Victoria Hospital. It is now manufactured by the Storz Instrument Co., 4904 Laclede Ave., St. Louis.

Recently two cannulas have come to my attention, one blunt, with more of a downward curve at the end,[15] and the other a straight, sharp instrument with a vertical pin near the tip.[16] Quite by accident these two main features have been incorporated in the cannula suggested.

SUMMARY

There are two methods of irrigating the sphenoid sinus: (*a*) through the natural ostium and (*b*) through puncture of the thin anterior wall of the sinus.

Making an opening in the anterior wall has certain advantages; material aspirated in this way is less liable to be contaminated, and irrigating fluid is syringed through more easily if there is free exit through the natural ostium.

A sphenoid cannula is suggested and the method of introduction described.

Preliminary roentgenograms are recommended, particularly the lateral view, to determine the level of the cribriform plate and the dimensions of the sphenoid sinuses.

A method of ascertaining the level of the cribriform plate by means of a spectacle frame is suggested.

This frame is also useful in many nasal operations.

1390 Sherbrooke Street, West.

15. Laurens,[8] p. 576.

16. Lobell, A.: Puncture-Irrigation of the Sphenoidal Sinuses with a New Instrument, Laryngoscope **36**:270-282 (April) 1926.

MECHANISM OF EFFECT OF ESTROGEN ON NASAL MUCOSA IN ATROPHIC RHINITIS

Successful Treatment with Prostigmine Methylsulfate; A Further Report

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The local treatment of atrophic rhinitis with estrone has been described by Mortimer, Wright and Collip.[1] The use of this substance for the treatment of

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1. Mortimer, H.; Wright, R. P., and Collip, J. B.: Canad. M. A. J. **37**:445, 1937.

ozena was suggested to them by their observations of periodic changes in the appearance of the nasal mucosa during menstruation. Oily solutions of estrone were repeatedly sprayed onto the nasal mucous membranes, and clinical improvement was reported. Blaisdell,[2] noting a similarity in the pathologic changes of atrophic rhinitis and of atrophic vaginitis, arrived at a like idea for the treatment of this entity. He also reported clinical improvement achieved through intranasal estrone therapy.

One of us (S. S.) made an endocrinologic study of a number of patients with advanced atrophic rhinitis and failed to find evidences of the endocrine disturbances described by Collip and his co-workers. Furthermore, treatment of these patients with intranasal estrone produced only mildly favorable or negative results. These two facts depreciated the role of endocrine disturbances in this form of nasal disease but did not explain such therapeutic results as were obtained through the use of estrone. However, a recent study by one of us,[3] revealed that the action of estrone may be classified into two categories: (1) the specific proliferative effect and (2) effects secondary to hyperemia. The latter effect appeared to us to be the important lead in explaining the results of treatment with this substance on the clinical course of ozena.

Reynolds [4] demonstrated that the hyperemic effects of estrone are due to the acetylcholine-liberating properties of this factor. This fact suggested that the benefit derived from intranasal estrone therapy in atrophic rhinitis depended not on a specific gonadal action but on incidental hyperemic effects that the hormone had on the nasal mucosa. A mode of therapy directed toward physiologic hyperemia without exposure to possible deleterious hormonal effect immediately suggested itself. The direct use of acetylcholine was avoided, as it was felt that the use of this substance was not entirely safe. The action of the naturally occurring acetylcholine in the nasal mucosa is counterbalanced by that of naturally occurring cholinesterase. A synthetic physostigmine-like substance which is commercially available has this specific action, and our procedure was to teach the patients with atrophic rhinitis to spray into each nostril four times a day 0.25 cc. of prostigmin methylsulfate in a solution of 1 to 2,000.[5] A preliminary report of the results of this treatment has been published.[6] The purpose of the present communication is to analyze the clinical course of a group of patients treated with this form of therapy.

Twenty patients were treated for periods varying from one month to one year. All other therapy was stopped except irrigation of the nose once or twice daily, to insure good contact of the medication with the mucous membrane of the nose. Ozena was controlled in all instances, usually at the end of the second week. Crust formation was definitely decreased and in some instances disappeared altogether. The nasal and pharyngeal mucous membrane became less dry and glazed, so that the discomforts resulting from pharyngitis and laryngitis sicca were greatly relieved. There was a definite reduction of space in the nasal vaults, but in no patient was there return to normal limits, except in 1 who was treated when the

2. Blaisdell, I. H.: Laryngoscope **48**:669, 1938.

3. Hechter, O.; Lev, M., and Soskin, S.: Endocrinology **26**:73, 1940.

4. Reynolds, S. R. M.: J. Physiol. **95**:258, 1939.

5. The prostigmin methylsulfate was supplied by Hoffmann-La Roche, Inc., through the courtesy of Dr. R. B. Shaner.

6. Soskin, S., and Bernheimer, L. B.: Mechanism of Estrogen Effect on Nasal Mucosa in Atrophic Rhinitis; Successful Treatment with Prostigmin, Proc. Soc. Exper. Biol. & Med. **42**:223, 1939.

disease was in an early stage. In all the other 19 patients the disease was advanced and of many years' standing. Subjectively all 20 patients were greatly improved.

Three of the patients stopped treatment for four weeks. In each instance, crusting, ozena and pharyngitis sicca again manifested themselves, so that it was necessary to resume treatment. The latter observation indicates that the beneficial results are not permanent if treatment is stopped. However, the patients are uniformly of the opinion that the treatment is eminently worth while, even if the treatment must be continuous. No untoward results have been observed from the early use of the drug in the specified amounts.

CONCLUSIONS

From the observations presented the following conclusions may be drawn:

1. The therapeutic results obtained in the treatment of atrophic rhinitis with estrogen are not the direct result of an endocrine action but are due to the effects secondary to local hyperemia.

2. Prostigmine methylsulfate applied directly to the nasal mucous membrane produces hyperemia by inhibiting cholinesterase.

3. Excellent results in the treatment of atrophic rhinitis have been obtained by the direct application of prostigmine methylsulfate to the nasal mucous membrane.

PREPARATION OF A PHYSIOLOGIC SOLUTION FOR NASOSINAL TREATMENT

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Ventilation of the nose and sinuses by vasoconstriction alone has become a recognized and widely used method of treatment. For acute and subacute infections it appears to be physiologically justified. To those convinced of its usefulness the following formula is recommended:

1. Water + ephedrine sulfate to 1 per cent + sodium chloride to 0.68 per cent.
2. Boil.
3. Keep in boiled, screw-capped bottles.

Either distilled or tap water may be used. If distilled water is used the solution will need to be alkalinized and buffered at or near the physiologic p_H, 7.6; if tap water, the p_H may or may not need adjustment. The use of tap water was suggested by Dr. Clifford Sweet[1] several years ago. It has proved to be exceedingly advantageous. Natural drinking water usually is alkaline, and the alkalinity is reduced somewhat by the addition of ephedrine. For example, in the San Francisco Bay area the use of tap water results in a p_H so near to normal that no adjustment is needed. It is not unlikely that this may be rather generally true. In some instances tap water should be submitted to Berkefeld filtration.

In the production of osmotic pressure 1 per cent ephedrine is equal to 0.17 per cent sodium chloride.[2] Hence, if dilution of the foregoing solution is desired it should be made with an aqueous solution of 0.85 per cent sodium chloride.

1. Sweet, C.: Personal communication to the author.
2. Schmidt, C. L. A.: Personal communication to the author.

Ephedrine solution made according to this formula is suitable both for atomization and for lateral head-low postural treatment.[3] For postural treatment it should be warmed to near body temperature.

Certain very desirable features characterize this solution:

1. Values for osmotic pressure and hydrogen ion concentration, physiologic.
2. Sterile, yet contains no antiseptic.
3. Stable in physical characteristics and effectiveness.
4. Odorless, colorless, clear.

The presence of this solution in the nasal fossae causes no sensation. This is significant in tending to confirm physiologic compatibility. Clinically it is fortunate, because it permits use for patients of all ages and sensibilities without causing complaint.

Moderately rapid vasoconstriction follows its local application in appropriate cases. The ventilation is reasonably prolonged, depending, of course, on the type and degree of pathologic change.

No compensatory vasodilatation has been observed. No local reaction has been encountered. This is credited to the physiologic compatibility of the solution. Experience is convincing that ephedrine in a perfect vehicle is free from significant side effects. Side effects appear to come solely from errors in the vehicle. The commonest errors are hypotonicity and acidity and also occur in the use of antiseptics and oils. The prevalence of imperfections in ephedrine solutions has led to the erroneous assumption that the drug itself has undesirable local effects. This is a case in which a crime undoubtedly has been committed but an innocent agent accused.

General evidence of ephedrine drug action does not accompany intranasal application performed correctly. Occurrence of general ephedrine effect is evidence of faulty technic, in that some of the solution has entered the pharynx and has been swallowed. Nasosinal absorption alone is insufficient to give significant general effect, but absorption from the gastrointestinal tract is virtually complete.

In making a physiologic solution for intranasal use, the aim is to approximate extracellular fluid. The guide is the cardinal physical characteristics common to both cellular and extracellular fluids. These have to do with specific values for osmotic pressure, hydrogen ion concentration and temperature.[4] Accuracy is essential if nontraumatic vasoconstriction is to be obtained. Yet, as small errors are likely, if not inevitable, it is necessary to know in which direction the greater safety lies.

Nasal secretion, derived from extracellular fluid, originates with physiologic values for osmotic pressure and hydrogen ion concentration. But change takes place quickly. Evaporation of water during inspiration causes concentration of the solute, with consequent increase in osmotic pressure (hypertonicity). It would appear, therefore, that nasal epithelium has a normal tolerance for slight hypertonicity. Hence, error in osmotic pressure should be toward the hypertonic side.

The situation in regard to hydrogen ion concentration is somewhat similar. Determinations of the p_H of nasal secretion show a range of from 6.5 to 8.5, whereas body fluids have a constant value of 7.6. This suggests mucosal tolerance of slight error in either direction. However, error in the p_H of the solution prob-

3. Parkinson, S. N.: A Lateral Head-Low Position for Nasal and Sinus Treatment, Arch. Otolaryng. **17**:787 (June) 1933.

4. Gamble, J. L.: Extracellular Body Fluid, Internat. Clin. **2**:184 (June) 1936.

ably should be toward the alkaline side, because absorption of carbon dioxide from exhaled air tends to increase the hydrogen ions. Were the solution prepared with acid reaction, it would be less likely to shift toward normal, as there seems to be no factor regularly present to decrease the hydrogen ion concentration.

According to available facts, nothing less perfect than a physiologic, aseptic solution should be used on inflamed nasal or sinal epithelium.

The principles used as a guide in making a physiologic solution of ephedrine apply with equal force to the use of synthetic vasoconstrictors. Choice should be limited to those which are stable. Obviously stability is essential in a physiologic solution.

The utility of vasoconstriction in rhinologic treatment cannot be fully appreciated until one has used a physiologic solution free from all side effects. It replaces virtually all the chemically and physically traumatic forms of treatment now and formerly in vogue.

Dr. Gertrude Moore, pathologist, verified both theoretic and practical features of this work.

Progress in Otolaryngology

Summaries of the Bibliographic Material Available in the Field of Otolaryngology

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PURULENT OTITIS MEDIA, MASTOIDITIS, SINUS THROMBOSIS AND SUPPURATION OF THE PETROUS PYRAMID

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A great part of the civilized world is at war. The source material of foreign origin published in 1939 is indeed meager. Surveying that which is available, one is struck with the absence of intellectual efforts to study definite phases of pending otologic problems. Contributions to the literature within the limits of my topics seem mostly concerned with case reports. The writers content themselves with recording observations without particular reference to their significance as influencing previously held concepts, nor is there much effort observable to establish newer concepts. It may be stated that the catastrophe which is afflicting humanity has considerably dampened scientific inquiry and study. Obviously lacking the necessary environmental serenity for thinking medical problems through, observers record "findings," leaving correlation and deduction from them to some one else, working in a happier time. That men of science are mentally preoccupied with the political fate of their countries must be appreciated when one is confronted with the paucity of sound scientific contributions in the year under review. Finally, medical reports from the medical services of the various armies are as yet unavailable. The trauma to the labyrinth caused by dive-bombing, plunging airplanes, the trauma to the middle ear from explosives and the effects on hearing of long submersion in submarines are topics which naturally cannot be expected to be on record at this time. All these need notation here, lest a misconception obtain regarding the quality of the material which is embraced in my report.

Sulfanilamide therapy still is a topic of considerable discussion. Sulfapyridine and sulfathiazole are newer therapeutic agents which are proving successful mediums for combating infections from the pneumococcus and the staphylococcus.

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LABORATORY METHODS

The preparation of vaccines from bacteria is a well established laboratory procedure. No attempt had been made heretofore to produce and use an antifungus. Mood [1] has done this, and his paper contains specific directions for the preparation of this material. He terms it an autogenous vaccine. The correctness of the term as applied to fungi may be questioned, yet the important factor in his report remains. He successfully treated with his "autogenous vaccine" 32 patients who were infected by various forms of Aspergillus in the external auditory canal. Since fungous infections of the external auditory canal often are troublesome and sometimes are difficult to control and heal, Mood's method is a useful addition to otologic therapy. He reports that in cases in which there were recurrences, resumption of the vaccine therapy resulted in cure of the lesion.

Frenckner [2] studied the rate of sedimentation in 1,341 cases of otitis media and its complications, employing both microscopic and macroscopic methods. The tests were repeated every second day during the illness. In cases of chronic purulent otitis media, there was no rise in the sedimentation rate. A slight rise was noted in cases of sinus thrombosis. There was no rise in the rate of sedimentation in cases of purulent labyrinthitis, while there was a slight rise during the late stages of meningitis. In cases of simple acute otitis media there was a slight rise in the sedimentation rate during the first week; in cases of purulent acute otitis media there was a definite rise during the first week In other words, there seems to be a rise proportionate to the severity of the infection. Frenckner states that if in the case of an adult with purulent acute otitis media the rate of sedimentation shows a rise of 100 or more, mastoiditis is present. In cases of otitis media in children, this does not always hold true. When the rise persists for more than two or three weeks, Frenckner believes, surgical intervention is indicated. If after operation the increase in the sedimentation rate is maintained or rises still further, this condition is considered to signify that some other focus of infection in the temporal bone exists, especially in the pyramidal apex. Frenckner holds that these sedimentation findings are present even before roentgenograms evidence a pathologic condition of the petrosal pyramid.

There is considerable in this report which is open to question. My own experiences with the use of sedimentation tests as a diagnostic aid have been unsatisfactory. True, the tests gave evidence of the presence

1. Mood, G. McF.: Specific Autogenous Vaccine Treatment of Otomycosis: Preparation of Vaccine, Am. J. Trop. Med. **18**:703, 1938.

2. Frenckner, P.: Sedimentation Reaction in Otitis Media and Its Complications, Acta oto-laryng. **26**:309, 1938.

in the body of infection, but the results were in no way specific enough to be of practical diagnostic use. Taking Frenckner's report at its face value, it is difficult to comprehend why labyrinthitis and sinus thrombosis should be accompanied with such meager sedimentation values, while a pathologic condition too slight to give roentgen evidence of its presence should cause a reaction of diagnostic value; nor do I believe that the severity and extent of an infection of acute otitis media can be estimated by the range of rise in the sedimentation rate. Too many other factors, for example the prior illness of the patient and the type of invading organism, play a role in activating the rate of sedimentation for this test to be used as an indicator for surgical intervention. The tests in my hands have been of value only as corroborating other laboratory and clinical findings.

THE ACUTE OTITIC LESIONS

Linck [3] raises an interesting question. He wonders why textbooks and other literature still maintain the differentiation and consideration of purulent otitis media and purulent mastoiditis as separate clinical entities, when in reality not only the air space comprising the middle ear but all the mastoid air spaces as well are usually simultaneously involved. In my own writing I have taken the same view, especially since the classic work of Schiebe and most of the roentgen studies of the mastoid process in the presence of tympanic infections have confirmed this. There is little present day logic in considering a purulent lesion as confined to the anatomic limits of the tympanum and the involvement of the mastoid process as a complication of the lesion of the middle ear. It becomes actually only a matter of the degree and intensity of involvement and the rapidity with which the intercellular bony walls succumb to the pressure factors inherent in the mechanics of coalescence.

Moura [4] observes that in children the presence of acute purulent otitis media often is overlooked because of the paucity of significant symptoms. He recommends a careful otoscopic examination in every case in which a child has an unexplained fever, loss of weight, meningismus or uncontrolled diarrhea. He recommends the performance of a myringotomy in such cases, even if the drum appears normal. To follow this recommendation blindly, I fear, would result in the incising of many normal drums, with the eventual development of infection of the middle ear. I believe every effort to make a diagnosis should be exhausted before exploratory myringotomy is undertaken.

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3. Linck, A.: Die akute Otitis media ohne "Mastoiditis," Arch. f. Ohren-, Nasen- u. Kehlkopfh. **145**:174, 1938.

4. Moura, A.: Latent Otitis Media in Diseases of Children, Ann. de otol., rin. e laring. **4**:16, 1938.

Asherson [5] reports 2 cases of acute purulent otitis media, in both of which complete deafness suddenly occurred. Simple mastoidectomy having been performed, the return of normal hearing resulted. In still another case, when operation was delayed for eighteen days after the onset of deafness, the hearing remained impaired after operation. Asherson states that severe deafness in the early days of an attack of acute purulent otitis media is an indication for immediate surgical intervention.

The deafness which occurs suddenly in cases of acute purulent involvement of the middle ear and the mastoid process is usually associated with pneumococcic infections. The reactions of the tissues in the labyrinth take the form of a hydrops due to chemical reactions between the perilymph behind the membranes of the oval and the round windows, on the one hand, and the purulent contents of the middle ear spaces, on the other. The labyrinthine reactions, notably those from the cochlea, are not necessarily the result of actual invasion of the labyrinthine fluids by the organism. Promptness in the relief of this hydrops will help conserve the threatened auditory acuity; hence, I find myself in full accord with Asherson's recommendations, the factors present in the given case being those outlined by him. Too early operative intervention on the mastoid process, before reactionary limiting changes in the tissues have been set up by the pathologic process, carries with itself dangers of the subsequent development of complicating lesions. Hence the functional test of the auditory acuity in such cases should be carefully evaluated, particularly the hearing by bone conduction under conditions of masking —so as to establish definitely the fact that auditory acuity is lost or is progressively and rapidly growing poorer.

With the increase in aviation will come an increase in diseases of the middle ear. This may be expected all the more when flying in exceedingly high altitudes becomes more common. The conditions to which the nasopharynx and the ear will be subjected in these situations will approximate those with which the medical profession is already familiar from the lesions which workers with caisson disease present. It has been established that the predisposing factor in the production of aural lesions among such persons is a pathologic condition in the nose and the nasopharynx. De Gaulejac,[6] in a study of lesions of the middle ear among aviators, finds this to be the fact. Therefore, as a measure of preventive medicine, prophylaxis directed to remedy and remove pathologic conditions in the nose and nasopharynx is urged as an addition to other safety factors with which professional fliers should be provided.

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5. Asherson, N.: Severe Initial Deafness as a Sign of Acute Mastoiditis, Lancet **2**:946, 1938.

6. de Gaulejac, R.: Les otites moyennes des aviateurs, Ann. d'oto-laryng., 1938, p. 588.

Chavanne [7] reports the case of a 14 year old girl who had a bilateral myringotomy, which was necessary during an attack of grip. Soon thereafter, four large blisters appeared on the left auricle, followed five days later by facial paralysis on the left side. Bilateral simple mastoidectomy was done. Acute coalescent mastoiditis was diagnosed. Gradually the facial paralysis disappeared. Chavanne considers that the case presented two separate complications of the grippal infection, which were unrelated to each other; one complication was the mastoiditis and the other the herpes zoster. One could still further differentiate and say that the mastoiditis was the result of a bacterial invasion of the middle ear spaces by the causal agent of the grip, while the herpes zoster was caused by the effect on the nerve endings of the toxemia which the causative agent produced.

The Ramsay-Hunt syndrome is not unfamiliar to otologists, but its occurrence among the young is rare. Depetris and Sosa [8] report a case in which it occurred in a child 5 years of age. There were severe headache on the left side, a vesicular eruption over the left auricle and facial paralysis on the left side. As far as I know, this is one of the first instances of this syndrome reported as happening at such an early age.

Haiman and Adelman [9] report a case of mastoiditis due to Actinomyces. Recovery followed simple mastoidectomy. The clinical picture was that usually present in cases of acute mastoiditis. With few exceptions, due to characteristics inherent in the pneumococcus, the Friedländer bacillus and a few other specific organisms, the bone tissue reactions and the body reactions are the same, no matter which kind or type of micro-organism is the invading agent. In this case report, which describes a condition caused by Actinomyces, one should have expected a clinical picture of a chronic state. However, the disease ran an acute course. The thought remains that a concomitant bacterial agent might have been present but remained unidentified and overshadowed by the actinomycosis. Demianczyk [10] reports a case of primary actinomycosis of the middle ear occurring in a girl 14 years of age, in which the condition was mistaken for tuberculosis. A diagnosis of tuberculous meningitis was made. At autopsy, however, there was found actinomycosis of the right cerebellar hemisphere and a circumscribed fibrinous pachymeningitis in the region of the right tympanum, with caries of the right

7. Chavanne, F.: Mastoïdite aiguë bilatérale avec coexistence unilatérale de zona de l'oreille et de paralysie faciale, Oto-rhino-laryng. internat. **22**:193, 1938.

8. Depetris, P., and Sosa, A. M.: Zona otico y parálysis facial (sindrome de Ramsay Hunt), Arch. argent. de pediat. **9**:370, 1938.

9. Haiman, J. A., and Adelman, M. H.: Acute Actinomyces Mastoiditis, Ann. Otol., Rhin. & Laryng. **48**:95, 1939.

10. Demianczyk, E.: Primary Actinomycosis of the Middle Ear, Polski przegl. otol. **14**:292, 1938.

tegmen tympani. Serial section of the temporal bone showed that the actinomycosis had started at the eustachian tube, had invaded the middle ear spaces and from there had reached the posterior cranial fossa. Risch [11] also reports on actinomycosis of the ear. In his case acute mastoiditis, sinus thrombosis and arthritis developed. Even the blood culture yielded a growth of Actinomyces, in addition to a hemolytic streptococcus. In Risch's case, an adequate surgical procedure, transfusions and the administration of iodides resulted in recovery. Risch's article is valuable for its complete review of the literature on actinomycosis of the ear.

Another unusual infection of the middle ear spaces is reported by Oppenheimer, Dennis and Badeen,[12] who report on finding brucella infection. They report a case of a 26 year old woman who complained of intermittent pain in the left ear, which lasted for two months. This was accompanied with a progressive swelling of the cervical glands on the left side. There were no signs of involvement of the ear. Roentgenograms, however, demonstrated disease in the left mastoid process. During the period of observation the temperature rose, and anemia and leukopenia developed, which became progressively severe. At operation on the mastoid process a few drops of serous fluid was found, which when cultured yielded Brucella melitensis. After operation swelling of the glands disappeared rapidly. The convalescence was stormy, and granulopenia persisted for some time. Recovery eventually took place with administration of vaccine therapy.

Another unusual observation is that of Paunz,[13] who reports an extraordinary case in which an intracranial dermoid simulated acute mastoiditis. A girl of 5 years became ill with fever and moderate chills, which lasted two weeks. She suffered pain behind the ear, and a swelling developed over the right mastoid process. The auditory acuity became less. This clinical picture developed four weeks after an attack of chickenpox. Otoscopic examination disclosed a reddening of the drum head, and a swelling, which had the appearance of a subperiosteal abscess, was evident over the mastoid process. The child was somewhat stuporous at the time of operation. At operation a subperiosteal abscess, filled with greenish yellow masses, was uncovered. The squamous portion of the temporal bone was rough and gray in appearance. One and five-tenths centimeters behind the abscess, and above the external auditory canal, a perforation of the cortex was uncovered, through which cholestea-

11. Risch, O. C.: Actinomycosis of the Ear, Arch. Otolaryng. **29**:234 (Feb.) 1939.

12. Oppenheimer, A.; Dennis, E. W., and Badeen, M.: Involvement of the Mastoid and Granulopenia in a Brucella Infection, Pract. oto-rhino-laryng. **1**:305, 1938; abstracted, Zentralbl. f. Hals-, Nasen- u. Ohrenh. **31**:412, 1939.

13. Paunz, M.: Intracranial Dermoid Simulating an Acute Mastoiditis, Acta oto-laryng. **26**:729, 1938.

tomatous contents were observed. Further exposure yielded degenerated epithelial pearls and many hairs in bundles. The covering bone was very thin and reddened. The antrum was not opened. Recovery took place within two months. Histologic study showed young connective tissue with widened capillaries and hair. The dermoid evidently developed between the squama and the dura. Such tumors in this locality are exceedingly rare. Another case of a dermoid tumor is reported by Watanabe,[14] who found the growth in the mastoid antrum during a simple mastoidectomy for an acute suppuration. When the antrum was opened, an isolated grayish white and elastic tumor mass was uncovered, which was about the size of the tip of the little finger. Histologic examination showed that it was covered with squamous epithelium; its structure consisted of epithelial cells, sebaceous glands, hair follicles, hair and a large number of small cysts. Watanabe believes it to be a dermoid, derived from abnormal development of embryonic germinal tissue.

In a 20 year old patient there developed deafness on the right side, accompanied with severe chills and otalgia, which lasted for several days. Kanai,[15] who reports the case, performed myringotomy and recovered streptococci. Eight days later, at simple mastoidectomy, a coalescent mastoiditis was demonstrable, with an intact condition of the bony plate over the lateral sinus.

Five days later there developed a subcutaneous abscess below the mastoid wound and, successively, a series of such abscesses—one on the abductor side of the left arm and others in the rectus femoris muscle, in the quadriceps femoris muscle, in the soleus muscle and in the musculus hallucis longus. In all of these abscesses streptococci were found. The patient recovered. The stress which the author puts on the finding of an intact bony sinus plate leads to the suspicion that he reports this case because of the metastatic abscesses, which, he surmises, rose from an infection of the blood stream originating in the lateral sinus, and that in the presence of a coalescent mastoiditis he expected to find the sinus plate macroscopically perforated and the sinus wall exposed. In the average case this is the condition observed, but metastatic lesions can be carried by the blood stream from a primary bacteremia. The history of severe chills lasting several days before the mastoidectomy was sufficient to suggest that the illness during this period was due to an infection carried by the blood stream. The question of whether or not the lateral sinus was involved in this case and whether or not there had been a primary thrombosis of the jugular bulb coincident with the onset of the tympanic infection must remain unanswered, because the sinus was not explored and recovery took place. The subsequent develop-

14. Watanabe, T.: A Dermoid in the Mastoid Antrum, Otologia **11**:506, 1938.

15. Kanai, H.: Acute Mastoiditis with Polymyositis, Otologia **11**:511, 1938.

ment of the polymyositis may have contributed to the recovery, these abscesses acting as "fixation abscesses," which aided in cleansing the blood stream.

After studying 15 cases of measles in which there were acute infections of the ear, Meller [16] observes that when measles occurs in a patient who already is suffering from afebrile acute purulent otitis media the clinical course of the measles becomes markedly altered. There is a definite lengthening of the prodromal stage, and the febrile phase of the disease takes on the characteristics of a sepsis. Nor does the temperature show its habitual abrupt drop with the appearance of the rash; the high temperature persists for some time. Likewise, the so-called double crisis in the temperature is absent. Meller believes that in such situations the measles and the otitic involvement are unrelated conditions, which, however, influence each other, thus increasing the general severity of the clinical picture of the measles. These factors must be comprehended and weighed when one observes a persisting high temperature in a case of measles superimposed on a priorly existing afebrile acute purulent otitis media and must not necessarily be held to indicate that a serious complication is developing.

Urbantschitsch [17] reports 4 cases in which destruction of the cochlear apparatus followed the development of serous or purulent labyrinthitis during attacks of scarlet fever. In his cases the acute purulent otitis media came on in the early stage of the scarlet fever; in 1 case it occurred simultaneously with the rash and in another just before the appearance of the eruption.

In commenting on the report of these cases, I may add that the deafness which follows scarlet fever is often observed and that it is known to result from the toxemia produced by this disease. Within the bony framework of the cochlea there appears to be rapid development of a hydrops of the labyrinth, which, by the local pressure it exerts on the organ of Corti, destroys it; hence, the subsequent loss of hearing. In its varying stages the labyrinthine reaction to the toxemia has often gone unrecognized for what it signified, and the transient vertigo and dizziness, and even ataxia, have not been traced back to their labyrinthine significance by pediatricians, who generally treat patients with scarlet fever. If hearing is to be saved, the most prompt and energetic otologic intervention is necessary in such cases. The measure of therapy to be applied necessarily must differ with the condition which the given patient presents. It is beyond the scope of my report to dwell extensively on this phase. Suffice it to note Urbantschitsch's record and to comprehend

16. Meller, H.: Zur Klinik der im Verlaufe einer eiterigen Mittelohrentzündung auftretenden Maserninfection, Monatschr. f. Ohrenh. **72**:481, 1938.

17. Urbantschitsch, E.: Zur Frage der Ausschaltung des Innenohres bei Scharlach-Otitis, Monatschr. f. Ohrenh. **73**:1, 1939.

that progressively advancing deafness occurring in a case of purulent otitis media which is a complicating lesion to scarlet fever requires prompt remedial therapy, not only to abate the purulency of the middle ear but to conserve the acuity of the patient's hearing.

An interesting study is presented by Mesrin,[18] who reports on 4,666 cases of scarlet fever, in which 905 of the patients presented purulent otitis media. Of these, 79 required mastoidectomies. The necessity for surgical intervention depends, naturally, on the course and severity of the otitic infection, and these seem to be influenced by the season of the year in which the patient becomes sick. The percentage of aural involvements increases during the winter and the spring and decreases during the summer. I may remark in passing that the incidence of mastoidectomies in this report equals 0.087 per cent and the incidence of infection of the middle ear in the cases of scarlet fever equals 19 per cent. These figures must furnish interesting reading to the overenthusiastic advocates of sulfanilamide therapy for purulent otitis media. In the cases reported, sulfanilamide therapy, was not administered, yet such a low incidence of mastoidectomies is impressive.

A summary of opinions and observations is presented by Kanevsky,[19] who reports on acute purulent otitis media in cases of scarlet fever. He holds that the younger the patient with scarlet fever is, the more apt he is to have his exanthem complicated by acute purulent otitis media. The severer the attack of scarlet fever, the greater the possibility of the otitic involvement. In 62.3 per cent of the cases he studied, the ear was infected simultaneously with the appearance of the scarlet fever. Kanevsky finds that the early performance of myringotomy does not prevent the development of mastoiditis or stop the development of necrosis of the bone or the formation of sequestrums. He finds that mild mastoiditis appears most often between the sixth and the twentieth day of illness. Acute necrotic otitis appears within the first fifteen days after the onset of the scarlet fever. Sinus thrombosis occurs mostly in conjunction with the necrotic form of scarlatinal otitis.

Early surgical intervention and late intervention have their advocates, and the data each group presents has not tended to end the controversy. Jampol'skij,[20] basing his findings on the study of a large amount of clinical material, finds that the patients who came back for reoperation, requiring secondary mastoidectomies, and also those who died were usually from the group who were subjected to operation early in the

18. Mesrin, M. P.: Scarlatinal Otitis, Vestnik otol. **1**:52, 1939.

19. Kanevsky, V. L.: Acute Purulent Otitis in Scarlet Fever, Vestnik otol. **1**: 35, 1939.

20. Jampol'skij, H.: Chirurgische Grundsätze bei der Behandlung skarlatinösen Otitiden, Vestnik otol., 1937, no. 6, p. 618; abstracted, Zentralbl. f. Hals-, Nasen- u. Ohrenh. **31**:615, 1939.

course of the disease, that is, within three weeks after the onset of the otitis. He recommends that surgical treatment be undertaken as late as possible, when the lesion in the mastoid has localized. He excepts cases in which there are symptoms which indicate impending complications. I have always taught that too early intervention carries within itself inherent dangers, because of lack of localization and demarcation of the lesion within the boundaries of the mastoid process. Hence I find myself in accord with the observer on this point, and I can comprehend why his fatalities should be found in the group of patients on whom early operation was performed. The failure to secure healing and cure, however, I believe is due to the local devitalization of tissue which the scarlet fever carries with itself rather than to the factor of early operation. Naturally such patients require reoperation later, when the system has recovered and the rest of the body is again in a healthy state. While these patients generally are cured by the second operation, sometimes the lesions fail to heal even after reoperation. Eventually such patients may need some type of radical mastoidectomy before a cure can be effected.

Vaheri,[21] in studying the type of lesion produced by the clinical form of mastoiditis due to Pneumococcus type III, found that in 45 cases the usual coalescent type of mastoidal lesion was present 5 times. A subacute but progressive form of lesion occurred 25 times. The classic textbook picture of the typical "mucosus otitis" was seen 15 times. Vaheri prefers the term "latent mastoiditis" instead of the designation "mucosus otitis," since other organisms than the so-called Streptococcus mucosus can and do produce similar clinical pictures. I have so often commented on the misuse of the term "latent" as applied to an active infection in the mastoid process that I will not repeat my arguments here. I concede that similar clinical pictures can be produced by different micro-organisms; in fact, the tissue reactions which locally occur to produce the lesion are less dependent on any particular invading organism than on other local factors. Any invading organism which sets given factors into action locally produces tissue reactions; these reactions bring on a series of phenomena which is interpreted as the clinical picture. When any organism only slightly affects the local reactions of the tissues the clinical response by severe symptoms is meager. To this local phase of bacterial activity the term latency is inaptly applied. If the invasion progresses, severe symptoms occur when the tissues of the cranial cavity are reached; hence, it is understandable that in 104 cases of streptococcic meningitis, which Vaheri reports having studied, he observed this "latency," this period during which slight mastoidal symptoms were

21. Vaheri, E.: Ueber die durch Pneumococcus III hervorgerufene Mastoiditis, Acta oto-laryng. **26**:631, 1938.

evidenced. But an infection progressively advancing and eventually flaring up into a real meningeal infection was hardly "latent" while en route through the bone tissues of the mastoid process until it reaches the intracranial structures. Reporting on cases of Pneumococcus type III infection, Kökeny [22] reports 5 cases of mastoiditis caused by this organism. In 3 of these there were found perisinal abscesses. One patient had an extradural abscess, and in 1 phlebitis and meningitis developed and the patient died. Instead of employing terms which do not truly explain the lesion, Maybaum and Druss [23] group infections due to the pneumococcus type III according to their clinical course. This at least seems to be logical. These observers, studying 73 cases, found the clinical course prolonged and insidious in 18 cases. The usual clinical course they found present in 53 cases; a short clinical course, from two to five days between the onset and the presentation of symptoms of intracranial complications, was observed in 2 cases. They note the loss of auditory acuity and the absence of signs of tenderness over the mastoid and consider these important diagnostic factors. Among their 73 patients they report a mortality of 18 per cent. They recommend early paracenetesis, culture of the secretions from the tympanic cavity and, when operation is undertaken, complete evisceration of the mastoid cellular contents, and they place emphasis on the investigation of all possible routes of infection into the petrosal pyramid. These recommendations, standard as they are, cannot be too often stressed.

Giese [24] studied 27 cases of acute purulent otitis media in which inflammatory manifestations were present in the kidneys. He observes that when nephritis appears soon after the onset of otitis media purulenta acuta, the infection of the ear is the cause of the renal lesion. When the renal lesion appears late, after some weeks have elapsed since the onset of the aural infection, other causative factors than the condition of the ear should be sought. Occasionally the acute purulent otitis media follows acute nephritis. This is because of the patient's lowered resistance, resulting from the nephritis. In 16 of Giese's cases there was a simultaneous time of onset of the aural and the renal infection. He finds the cause of this twofold manifestation of general infection in some disturbance or lesion in the nasopharynx, the tonsils or glands or in erysipelas.

Hörbst [25] studied 350 cases of acute mastoiditis and reports on them. The mortality in these cases was 3.7 per cent. The roentgenologic diag-

22. Kökeny, D.: Mucosus Mastoiditis, Orvosi hetil. **83**:234, 1939.

23. Maybaum, J. L., and Druss, J. G.: Otitis Infections Due to the Pneumococcus Type III, Arch. Otolaryng. **30**:21 (July) 1939.

24. Giese, W.: Akute Mittelohrentzündung und Nierenentzündung, Arch. f. Ohren-, Nasen- u. Kehlkopfh. **145**:159, 1938.

25. Hörbst, L.: Zur Klinik der Mastoiditis, Monatschr. f. Ohrenh. **72**:580, 1938.

nosis was substantiated by operative findings in only 67 per cent. Hörbst states that he considers the most important element of the roentgen findings to be the knowledge they contribute of the distribution of the cells of the mastoid process. Sixteen per cent of the patients were subjected to operation within the first two weeks of the illness, 36 per cent between the third and the fourth week, 24 per cent between the fifth and the sixth week and 24 per cent even later. The invading organism was Streptococcus haemolyticus in the large majority of the cases; the pneumococcus type III was the next most frequently found organism. Half the cases occurred in the first quarter of the year. In 95 per cent of the cases the hearing became normal.

Woelk [26] does not believe that every patient with acute purulent otitis media which lasts six weeks should be submitted to mastoidectomy. He cites 1 case of six months' and another of eight months' duration, in which a pathologic condition in the mastoid process was not demonstrable at operation. He directs attention to the fact that roentgenograms, cultures of material from the ear and even the rate of sedimentation do not give clearcut indications for surgical intervention in such cases. He questions the benefit to auditory acuity from an operation undertaken solely on the consideration of the length of time acute suppurative otitis has been present. With this argument I find myself in accord, as the indication for surgical intervention must be based on more than the time element which the infection shows. There are other and more significant factors. Finally, it must be remembered that there are conditions which are chronic from the time of their onset. These chronic infections have inherent pathologic elements which, when recognized, place them in another category than that of an acute tympanic lesion, and they are therefore not included in Woelk's conclusions. Such conditions obviously would require a different type of surgical treatment than that comprised in the technic of the simple mastoidectomy. The simple mastoidectomy would not in most instances reach a pathologic condition such as those of which I am speaking.

Influenzal otitis presents a typical clinical entity. Roussel and Deyme [27] have studied this infection, and they recommend the intramuscular injection of 10 cc. of the patient's own blood for the prevention of the typical symptom complex of influenzal otitis. In cases in which the otitis is already established they report a sharp drop in temperature and a recession of all symptoms referable to the ear as a result of the use of this form of therapy. Finally, they recommend the injection as a prophylactic measure in epidemics of grip among soldiers, to prevent the development of influenzal otitis.

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26. Woelk, H. A.: Die Indikation zur Antrotomie bei lange bestehender akuter Otitis media, Hals-, Nasen- u. Ohrenarzt (pt. 1) **30**:51, 1939.

27. Roussel and Deyme: L'autohémothérapie dans la prévention des otites grippales, Bull. mens. Soc. méd. mil. franç. **32**:479, 1938.

Hirst [28] discusses the utility of douching the ears in cases of acute suppurative otitis media and states that he believes that douching the ears does not carry the infection into the mastoid cells. In experiments which I made years ago with staining fluids on cadavers in the membrana tympani of which I had made various sized openings, not only did I find that douching the ears as it usually is done does not bring the fluid into the mastoid cells, but I had great difficulty in making the colored fluid even pass the perforated ear drum. I was working on cadavers which had no pathologic fluids in the tympanic cavity. The conclusions I arrived at regarding the douching of the external auditory canal when the tympanic cavity is filled with purulent secretion under pressure and exuding through the perforated ear drum under the added pressure produced by each pulsation of blood vessels can therefore be surmised.

SULFANILAMIDE THERAPY

Horan and French [29] find that since they have instituted the use of sulfanilamide in the treatment of acute purulent otitis media the incidence of mastoiditis has fallen from 22.7 per cent to 4.5 per cent and that of fatal complications has dropped from 2.85 per cent to zero. These observers recommend that the administration of the drug should be started early in the course of the disease. They note that when mastoiditis does occur, the classic clinical picture is rarely observed and that operative treatment is resorted to late in the course of the disease. The latter observation is obvious because of the masking effects of the drug, which lull both patient and medical attendant into a sense of false security, until the ideal time for surgical intervention has passed. Because of this masking effect, my own practice during the last year in cases in which sulfanilamide was used has been to operate on patients who presented lesions less serious than those which I formerly deemed indicative of a surgical procedure. I hope that more experience will relegate the use of this valuable drug to the combat of serious septic lesions and will not popularize it for the treatment of simple otitic infections, because I believe its masking effects constitute a real danger and that serious lesions develop which are ordinarily preventable by any competent otologist.

Nevertheless, there is the opinion of Hubert [30] to the contrary. He finds, in his use of sulfanilamide for the treatment of acute purulent otitis media, that many myringotomies may be spared. He too warns of

28. Hirst, O. C.: Treatment of Acute Suppurative Otitis Media, Arch. Otolaryng. **30**:755 (Nov.) 1939.

29. Horan, V. G., and French, S. G.: Sulphonamide in the Treatment of Acute Mastoiditis, Brit. M. J. **2**:942, 1938.

30. Hubert, M.: Rôle des traitements sulfamidés dans la thérapeutique des suppurations auriculaires et leurs complications, Rev. de laryng. **60**:405, 1939.

its masking effects. I am fully in accord with Hubert in his recommendation to use the drug in cases of sepsis and of meningitis. Baker and Bradford [31] also report favorably on the results of the use of sulfanilamide. They report 4 cases which were, in their opinion, clinically cases of surgical mastoiditis. The 4 patients recovered without surgical intervention, after the use of sulfanilamide. Fisher [32] reports on 183 cases. Ninety-two patients were treated in the ordinary manner, and 88 were given sulfanilamide in addition to the usual form of treatment. In the group without sulfanilamide therapy there were 66 mastoidectomies; sepsis developed in 4 of these patients. The average time required for healing in this group of patients was sixty-five days. Mastoiditis developed in only 7 of the 88 patients who were given sulfanilamide; there were no complications, and the average time required for healing was twenty-three days. Houser [33] made a survey of the opinions of one hundred and six otologists and seventy-eight pediatricians, which yielded a response generally favorable to the drug. Houser criticizes the response to his survey by pertinently remarking that these responses were all made by observers who exercised no controls on the experimental use of the drug. It is hard to secure controls in this kind of clinical investigation. There are, in addition, seasonal variations and differing degrees of bacterial virulence. Before final judgment can be had on the use of this drug for ordinary mastoidal infections, much more time must elapse, during which observations must be carried on. The isolated reports which are detailed here have only a suggestive value. The incidence of mastoiditis for the year 1938-1939 was very low. Maybaum, Snyder and Coleman [34] made a report on which I commented last year. They again report and reiterate the masking effect of sulfanilamide on the classic course of acute otitis. They stress what I have already alluded to, namely, the false sense of security that is given to the patients and the physicians by the absence of symptoms and objective signs. They stress that evisceration of the focus of infection is of paramount importance. They contend that the drug obscures diagnosis and results in a masked or latent involvement of the mastoid process—thus modifying the course of what ordinarily would be a clearcut classic picture

31. Baker, D. C., Jr., and Bradford, G. E.: Sulfanilamide Therapy for Acute Otitis Media and Acute Mastoiditis, Arch. Otolaryng. **29**:334 (Feb.) 1939.

32. Fisher, G. E.: Sulfanilamide in the Treatment of Otitis Media, J. A. M. A. **112**:2271 (June 3) 1939.

33. Houser, K. M.: A Survey of the Use of Sulfanilamide in Acute Otitis Media, Laryngoscope **49**:825, 1939.

34. Maybaum, J. L.; Snyder, E. R., and Coleman, L. L.: The Value of Sulfanilamide in Otogenous Infections with Special Reference to Its Masking Effect, J. A. M. A. **112**:2589 (June 24) 1939; Experience with Sulfanilamide Therapy for Otogenous Infections with Specific Reference to Masking of the Clinical Course, Arch. Otolaryng. **30**:557 (Oct.) 1939.

of mastoidal disease. Nevertheless, in spite of the administration of sulfanilamide, patients still come to operation, but often when they do they present complications. As I stated in last year's comment, what is actually needed is a new conception of the indications for operation in cases under sulfanilamide therapy. This involves a different interpretation of the clinical laboratory data as well as a different reading of roentgen films. The better radiologists have begun to appreciate the latter factor, and reports are commencing to be received which state the degree of decalcification, rather than the negative reports which were wont to be made a year ago. Eventually, when these elements are fully mastered, sulfanilamide will be used without the threat its employment now holds.

SURGICAL TECHNIC—ENDAURAL METHOD

Recently in certain scattered spots in this country a veritable campaign seems under way to popularize endaural surgical procedures. The reasons for this are incomprehensible, yet it is going on in some of the better institutions, in spite of the fact that the standard simple mastoidectomy, of all types of surgical intervention of which I am aware, probably ranks highest as a life-saving measure. The use of the endaural route has been advocated in Europe for nearly two generations without much real progress toward its general adoption as a routine procedure. For me, who am wont to survey current literature and to study case reports in order to note trends and advances, who have some experience with both methods of surgical technic and who also have had ample opportunity to see the end results from both types of procedure over a course of years, one fact stands out which appears incontrovertible: *There is no scientific factor sacrificed by the use of the standard postauricular route of operation which is saved by the employment of the endaural route.* The claim advanced for the use of the endaural approach, that there is total absence of a scar after operation, can be made only to an unintelligent class of patients. There is a scar, and it is more visible on the external auricular surface than is the usual scar behind the auricle and covered by it, which results when the incision is made near the attachment of the auricle to the skull, in the more usual operation.

It is difficult to escape the thought that endaural surgery, because of its novelty here in America, excites the curiosity and challenges the skill of the clever surgical technicians, who seem to get some sort of thrill out of performing a difficult surgical feat successfully. Furthermore, in this age of keen competitive private practice, the claim advanced to the layman that an operation will be performed "without cutting into the skin from the outside" is a "salesman's" argument and does not indicate the selection of a definite surgical procedure necessitated by the

presence of specific pathologic conditions. Nevertheless, it must be recognized that this argument holds a fascination for the ignorant layman, who, by implication, is led to believe that he is undergoing a less dangerous operation "because there will be no incision," and "all the work will be done inside the ear." Those of us who know anything about this phase of surgery know better than to accept these premises. The patient runs exactly similar risks in the use of the endaural and of the postauricular method of entering the mastoid process surgically.

Wishart,[35] who recently made a study of endaural surgery, reports his opinion anent it. He quotes Whiting's standard requirements for a successful mastoidectomy. These are "complete eradication of all diseased bone, in the shortest time commensurate with thoroughness, by methods designed to reduce to a minimum the dangers incident to the operation and calculated as well to induce speedy and permanent healing." Wishart feels that in the choice of the endaural route of operation the surgeon does not follow these recommendations as completely as he does in the choice of the standard postauricular route, and, consequently, the former is not a method of choice for the simple or for the radical mastoidectomy.

Wishart draws attention to some specific disadvantages of the use of endaural surgical treatments for the ear, particularly as this type of surgical procedure has been modified by the incisions devised by Lempert. He directs attention to the possibility of introducing infection into the temporal region and states as his opinion that the procedure gives inadequate exposure of the tip and of the postsinal regions. He fears that damage may be done by the necessary use of retractors introduced into the incisions into the concha and the canal wall, with the resultant development of perichondritis. He reports that the postoperative wound is more sensitive than that resulting from the standard operation. The period of healing, the period of operation and the patient's stay in the hospital are prolonged over those required when standard procedures are employed. Wishart draws attention to objectionable, conspicuous scars with keloid development which result in some cases, and he reports on the occurrence of pear-shaped contracture formations, which distort the external auditory canal, as an end result in some instances. This observer states that the more abnormal the condition of the superficial tissues is in a case of acute mastoiditis, the greater is the difficulty of inspecting the depths of the cavity.

On the other hand, Howarth and Bateman,[36] who used the endaural approach for simple mastoidectomy in 20 cases, favor this type of proce-

35. Wishart, D. E. S.: The Endaural Route, Laryngoscope **49**:943, 1939.

36. Howarth, W., and Bateman, G. H.: The Endaural Approach to the Mastoid, Lancet **2**:1168, 1938.

dure. In 3 cases they employed the endaural approach to perform a radical mastoidectomy, and 7 times they used it for modified radical mastoidectomies. Reporting on their experience with these 30 cases, they consider this method of approach better than that of the usual postauricular opening into the mastoid process.

CHRONIC CONDITIONS

It is some time since a report on the bacterial infection present in a case of chronic mastoiditis has come to hand. Today physicians are wont to hold that the bacterial flora incidentally present in the chronically suppurating ear is not the causative element in the basic pathologic condition which presents itself in chronic mastoiditis. Therefore, the case report of Newhart [37] is interesting and important. In a patient 32 years of age, who had had chronic purulent otitis media on the left side since he was 4 years old, the discharge from the ear suddenly became bloody and otalgia developed. Within sixteen days a chill, sweating and vomiting presented; these symptoms were followed by an icteric condition of the sclera, a spiking type of temperature curve, rigidity of the neck, a positive Kernig sign and pain in the chest. During the period of observation, the blood culture yielded anaerobic hemolyzing colonies of long, threadlike bacilli with purple granules, which were identified as Bacillus fragilis. At operation a large cholesteatoma was found, which was accompanied with an old perisinal abscess, and a sinus obliterated by nature in the process of the development of the lesion. Pus from the mastoid also gave B. fragilis. The sinus was thrombosed and, where not obliterated, contained thin, dark, foul-smelling pus. Despite ligation of the internal jugular vein and other treatment, the patient died. At autopsy a cerebellar abscess was found on the right, the side opposite the aural lesion. There were present also meningitis, old thrombi in the superior sagittal and petrosal sinuses and two small abscesses in the liver. Obviously it would take me too far afield to make comments on the many interesting problems which this case presents—a twenty-eight year persistence of chronic mastoiditis, with cholesteatoma; the infection of the blood stream, with metastatic foci in the brain, on the opposite side to the aural lesion, and in the liver; the protective tissue reactions which closed off and spontaneously obliterated part of the sinus and the old thrombi in other blood sinuses—but let it suffice to draw attention to the rarity of the infecting agent during the terminal phase of the patient's illness. B. fragilis is identified and can be isolated only as an anaerobic culture. The necessity for the employment of such methods of culture in cases in which aerobic culture mediums fail

37. Newhart, H.: Report of Unusual Bacterial Findings in a Fatal Case of Chronic Otitis Media with Complications, Laryngoscope **49**:1024, 1939.

to reveal bacterial growth is obvious. It must be noted, too, that the twenty-eight year duration of the suppuration of the middle ear is to be considered as due not to the presence of B. fragilis but to the epithelial ingrowth—the cholesteatoma.

Madge [38] makes an interesting report on the results of transmastoidal drainage for the treatment of chronic suppurative otitis media in children. He treated 103 patients, who had what he terms suppurative otitis media, by performing a simple mastoidectomy and then washing through the middle ear and the open antral spaces; this procedure was followed by the application of zinc ionization. One group in whom the suppuration had persisted for only from six weeks to three months is also included among those submitted to combined surgical therapy, irrigation and ionization. This group comprised 27 patients, of whom all but 1 were healed. Of course, I have always contended that chronicity has no relation to the element of time and that a chronic condition can often be diagnosed almost from the time of the onset of the otorrhea; but Madge does not give the diagnostic data by which he classified the infections of short duration as chronic. Of his second group of patients, the duration of whose symptoms was from three to six months, 11 were healed and 18 were improved. The third group is composed of patients with symptoms of more than six months' duration. Of these, 3 were healed and 18 were improved. Madge notes that in the patients of the first group healing often occurred spontaneously. The futility of attempting to secure cures for chronic conditions involving the tympanic cavity, the aditus and the mastoid antrum by simple mastoidectomy is evident from the poor results reported in his last group of cases. The truth is again apparent that inadequate surgical treatment, which cannot reach the seat of the lesion and which fails to remove the lesion, is a futile gesture.

Radzyminski and Krajnik [39] studied the histopathologic picture presented by polyps of the middle ear occurring in 676 cases. In the majority of cases these polyps have their origin in granulations in the tympanic cavity. Six per cent of the polyps were found during the first year of life, 8.7 per cent in the period up to 10 years of age and 19.1 per cent in the second decade of life, 17.6 per cent in the third, 16.4 per cent in the fourth and 9.6 per cent in the fifth.

On a patient 45 years of age, who had suffered from chronic purulent otitis media for six years, Kecskes [40] performed a radical mastoidectomy

38. Madge, F. T.: The Results of Transmastoid Drainage of the Middle Ear for Chronic Suppurative Otitis Media in Children, J. Laryng. & Otol. **53**:711, 1938.

39. Radzyminski, A., and Krajnik, B.: Ueber histopathologische bilder von Mittelohrpolypen, Polski przegl. otol. **13**:98, 1937; abstracted, Zentralbl. f. Hals-, Nasen- u. Ohrenh. **31**:375, 1939.

40. Kecskes, Z.: Sequestration of the Cochlea in the Course of a Chronic Purulent Otitis Media, Orvosi hetil. **83**:65, 1939.

without giving the patient relief from headache or from the persistent otorrhea. Three months later a reoperation was undertaken, and a sequestrum was removed, which comprised the entire structure of the cochlea. Thereafter rapid healing ensued, and the patient was cured.

Podesta [41] reports a rare finding. Cholesteatomas generally are found in patients with chronic conditions associated with sclerosis of the mastoid bone. However, he presents a case in which there was a cholesteatoma in a well pneumatized mastoid process, wherein the matrix had penetrated and spread into the cells, some of which were far removed from the mastoid antrum. Of course, those who comprehend the mechanics of development of a cholesteatoma know that an ingrowth of pavement epithelium can possibly happen in any type of temporal bone structure. The rarity is not so much that a cholesteatoma developed in this type of bone, for buried squamous epithelium almost always will produce what is termed a cholesteatoma, but that a chronic condition should develop in a pneumatized bone and should have the inherent factor which favored the ingrowth of epithelial tissue. Van Caneghem [42] distinguishes between cholesteatoma of the external auditory canal, with accumulation of cerumen and epithelial desquamation, and cholesteatoma of the middle ear, which grows into the external auditory canal. Of 24 cases, in 14 the condition was bilateral and in 10 unilateral. Of 38 ears studied, 21 presented perforation of the membrana tympani, while 15 showed perforations in the shrapnellian region. In 6 cases the perforation was within the boundaries of the pars tensa. In 17 cases no perforation existed. A noteworthy observation by the author is that all these patients suffered from intractable cough. The cause of this cough, according to the author, was chronic infection of the paranasal antrum and the ethmoid sinuses. He theorizes as to the causation of the infection and traces a chain of causative factors: First there is the production of a desquamative hyperkeratitis, which is secondary to a disturbance of nutrition, this, in turn, being due to a chronic infection in the rhinopharynx. Mayer [43] also reports on cholesteatoma. He studied 4 cases in which there was cholesteatoma of the external auditory canal. One was that of a person with congenital syphilis who had a gumma and also a cholesteatoma in the external auditory canal. In his second and fourth cases there was a sequestrating periostitis present with a cholesteatomatous formation in the canal. In all 4 cases, periostitis with necrosis of bone was found, and there were large cavities in the bone of the external auditory canal, which

41. Podesta, R.: Sobre una de las particularidades de los colesteatomas del oido, Rev. oto-laring. de São Paulo **6**:133, 1938.

42. van Caneghem, D.: La cholestéatome du conduit et la pathogénése du syndrome, Ann. d'oto-laryng., 1938, p. 399.

43. Mayer, O.: Ueber die Entstehung des Cholesteatoms des aüsseren Gehörganges, Hals-, Nasen- u. Ohrenarzt (pt. 1) **29**:283, 1938.

were filled with epidermis. The role of the epidermis here, according to Mayer, is a passive one, in contradistinction to its function in cases of cholesteatoma of the middle ear and mastoid process, wherein it assumes an active role in the production of the lesion. In 3 of his cases syphilis was the causative factor. Mayer believes that tuberculosis can produce a similar lesion.

In my survey last year, I alluded to the use of urea in the treatment of chronic purulent otitis media, and in the literature under review this year there appears another report on this topic. Tremble [44] combined urea and caroid, both in solution and in a powdered form, and obtained good results in the treatment of chronic purulent otitis media. When used in solution, as Tremble employs it, the strength of urea is 8 per cent to 10 per cent; that of caroid is 10 per cent. He finds that the solution dissolves debris and necrotic tissue. It should be instilled into the ear through the eustachian tubes by means of a catheter.

TECHNIC OF RADICAL MASTOIDECTOMY

Goodyear [45] comments on the advisability of effecting a closure of the eustachian tubes in all radical mastoidectomies, because the open tube permits nasal secretions due to a cold to find their way into the cavity left by the radical mastoidectomy. He therefore devised a procedure to be added to the usual technic of operation. After the exposure of the middle ear the mouth of the eustachian tube is widened and exposed, and the lip of bone overhanging the tube's mouth is removed. The mucous membrane is thoroughly curetted out from the tube. The tensor tympani muscle is then identified in its course along the inner tympanic wall. The muscle is elevated from its bed and tucked into the tube. Goodyear describes in detail the technic he advocates. He states that with this procedure he successfully closes the eustachian tube.

To my mind the absolute necessity of producing a closed eustachian tube in a radical mastoidectomy is still a moot question. The widely held belief to the contrary notwithstanding, I question the theory that when the body undergoes what is commonly called a "cold" there is a flow of fluid from the nasopharynx through the eustachian tubes into the mastoidectomy cavity. I do not desire to open this question now. Nevertheless, while directing attention to Goodyear's proposals and his reasons for making them, I desire my readers to remember that when an operative cavity is healed and the patient is cured, if fluid escapes through the eustachian tubes into the healed cavity because a "cold" has subsequently developed, the fluid will find itself lying on healthy

44. Tremble, G. E.: The Use of Urea and Caroid in Chronic Otitis Media, Canad. M. A. J. **40**:149, 1939.

45. Goodyear, H. M.: The Radical Mastoid Operation: Use of Tensa Tympani Muscle in Closing the Eustachian Tubes, Laryngoscope **49**:580, 1939.

squamous epithelium, which will remain as unaffected by this secretion as would the skin of any other part of the body with which it might accidentally come in contact. There are other factors, of entirely different import, which are brought into play when suppuration develops in a previously healed mastoidectomy cavity. I concede the necessity of keeping the mastoidectomy cavity as dry as possible during the process of healing. With these few comments this question must be left for the present.

INFECTIONS OF THE BLOOD CHANNELS

The surgical treatment of infections of the cranial blood sinuses has had its place in otologic literature for about fifty years. The indications for operation, the methods and the means for establishing diagnosis and even the surgical technic have been stabilized and considerably standardized. The moot question what to do about the internal jugular vein (whether to leave it intact, to ligate it or to resect it), presented to the profession by Lane in 1889, has not yet been answered satisfactorily. Finally, it must be remembered that, even with a resected internal jugular vein, it is surgically impossible to localize absolutely the infected blood streams. The vertebral and the condylar veins, and others of smaller caliber, are still able to carry infected material to remote parts of the body and to produce secondary septic foci.

A most interesting article comes from Woodhall,[46] who studied the anatomy of the cranial blood sinuses with particular reference to the lateral sinus. He is able to distinguish four main anatomic types of these sinuses: 1. The common pool type, wherein both the longitudinal and the straight sinuses empty into a common pool, from which blood is distributed equally to both lateral sinuses. 2. The plexiform type, in which the longitudinal and the straight sinuses divide and run largely into one or another side—resulting in some difference in the size of the two lateral sinuses. 3. What Woodhall terms the ipsilateral type, in which the longitudinal sinus runs to the left side, so that one lateral sinus is very large and the other one is small. 4. The unilateral type, which is rare and presents only one lateral sinus. When there is any blood vein present on the opposite side, it is much attenuated. Woodhall studied 100 skulls and found type 1 nine times, type 2 fifty-six times, type 3 thirty-one times and type 4 only once. He believes that many syndromes of venous stasis, otitic hydrocephalus and hemorrhagic encephalitis can be explained on the bases of these several variations in the anatomy of the cranial sinuses. Likewise, these venous variations may account for the difficulty at times encountered in interpreting the results of the Tobey-Ayer test.

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46. Woodhall, B.: Anatomy of the Cranial Blood Sinuses with Particular Reference to the Lateral, Laryngoscope **49**:966, 1939.

The rest of the available literature deals mostly with case reports. Thus Druss [47] studied 15 cases, including histologic examinations, and substantiates standard procedures based on classic indications. Giese [48] reports on spontaneous hemorrhage from the lateral sinus. During a period of fifteen years he had 5 such cases. In 3 the hemorrhage was due to a localized necrosis of the sinal wall, and in 2 it resulted from splinters of bone which traumatized the sinus. Three of the patients died.

Smith [49] brings another question into the discussion. The literature has on record 6 cases of bilateral sinus thrombosis and ligation of one jugular vein and 3 cases in which both internal jugular veins were tied. Smith reports the case of a 10 year old child on whom bilateral mastoidectomies had been performed. One week postoperatively sepsis developed, and at operation the left lateral sinus was found to be thrombosed. This sinus was obliterated, and the left jugular vein was tied. A week later, the sepsis continuing, the right sinus was opened and obliterated, but no thrombus was found. After this procedure hydrocephalus developed, and a metastatic lesion formed in the left elbow. The patient recovered. Calabresi [50] reports 2 cases which were asymptomatic. In 1, the thrombus was discovered at operation. No further surgical treatment was given except thrombectomy, and the patient recovered. The other patient, a 65 year old woman, on whom radical mastoidectomy was done to obliterate a cholesteatoma, died thirty-six hours later. At autopsy an aseptic thrombus was found in the lateral sinus. Koch [51] studied 34 cases of sinus thrombosis and tied the internal jugular vein in all. Fifteen of the patients recovered after the thrombectomy and ligation. In 10 cases no demonstrable thrombus was found, and the patients recovered. Nine patients died of metastasis and meningitis. Most of these were operated on very late in the course of the disease. Koch believes that ligation of the jugular vein should be carried out in all cases of otitic sepsis in which the disease condition continues, even though the sinus which is explored is found to contain blood. On the other hand, Traina [52] leaves the internal jugular vein

47. Druss, G.: Thrombosis of the Lateral Sinus: A Study of Fifteen Cases Including Histological Examination, Arch. Otolaryng. **29**:42 (Jan.) 1939.

48. Giese, W.: Spontane Blutungen aus dem Sinus nach Ohroperationen, Arch. f. Ohren-, Nasen- u. Kehlkopfh. **146**:23, 1939.

49. Smith, M. T.: Thrombophlebitis of the Lateral Sinuses Complicating Bilateral Acute Mastoiditis, Arch. Otolaryng. **29**:533 (March) 1939.

50. Calabresi, C.: Due casi di trombosi asintomatica del seno laterale di origine otitica, Arch. ital. di otol. **51**:129, 1939.

51. Koch, F. X.: Beiträge zur Jugularisunterbindung bei Sinuserkrankungen am Mittelohr, Monatschr. f. Ohrenh. **72**:672, 1938.

52. Traina, S.: Criteri clinici per la terapia chirurgica delle setticemie otitiche, Boll. d. mal. d. orecchio, d. gola, d. naso **56**:361, 1938.

alone in cases in which he finds a partial or a complete obliterating thrombosis of the lateral sinus without any evidence of sepsis. He exposes the sinus but does not disturb the clot. Only if symptoms appear does he operate. In cases in which sepsis is present he first exposes the sinus. If during several days' observation the temperature falls by lysis, he does nothing more. When, however, sepsis continues, Traina operates on the lateral sinus and on the internal jugular vein.

Joannovich [53] reports on 30 cases of sepsis observed at the Red Cross Hospital at Athens, Greece, in which 19 cures were obtained. In 12 of these the condition was caused by Streptococcus haemolyticus, and 11 of the patients recovered. Joannovich feels that the use of streptococcus serum of Vincent was largely responsible in bringing about this favorable percentage of recoveries. He had 7 patients in whom the sepsis was caused by the pneumococcus type III. Three of these patients recovered.

Iwaszkiewicz [54] reports on 26 cases of thrombosis of the sinus and the jugular bulb. In 20 of these thrombosis occurred in conjunction with chronic infections, and in 6 it was associated with acute lesions. In 14 the mastoid process was sclerotic, in 8 it was pneumatic and in 4 it was sclerodiploic. In some of the 16 cases in which the condition followed cholesteatoma only the sinus was involved, and in 10 cases the jugular bulb was also involved. Eight patients died. The causes of death were meningitis, thrombosis of the cavernous sinus, pulmonary infarct, endocarditis and pulmonary abscess, and pneumonia and cardiac failure were found in 4 cases. Cody [55] reports 8 cases with 4 deaths. The most important case in Cody's report is one in which a thrombosis of the cavernous sinus developed. The patient recovered after enucleation of the eye, drainage of the cavernous sinus and ligation of the internal jugular vein.

Klicpera [56] studied injuries to the blood sinuses occurring during mastoidectomies in the light of their seriousness. In a study of 20 cases of injury to the sinuses he found that in 17 there was normal healing, without any complications; in 3 cases septic sinus thrombosis developed and an additional surgical procedure became necessary. In these cases also the patients recovered. Klicpera holds that injury to the sinus occurring in a mastoidectomy is not as serious as it was formerly believed to be. He recommends that tamponade should be made over

53. Joannovich, D.: Therapeutische Resultate bei 30 Fällen von otogener Sepsis, Ztschr. f. Hals-, Nasen- u. Ohrenh. **45**:17, 1939.

54. Iwaszkiewicz, J.: Thrombosis of the Sigmoid Sinus and Bulb, Polski przegl. otol. **15**:611, 1939.

55. Cody, C. C., Jr.: Thrombosis of the Lateral Sinus, Arch. Otolaryng. **29**: 674 (April) 1939.

56. Klicpera, L.: Ueber operative Verletzungen des Sinus sigmoideus und ihre Folgen, Monatschr. f. Ohrenh. **72**:1059, 1938.

the site of the injury and that further clinical manifestations should then be awaited. In this regard it must be comprehended that when a septic condition of the sinus develops in the ordinary course of an infection of the mastoid process, the entire body is the seat of a reaction to the infection, which progresses to a degree and overcomes resistance, pending the time when operation on the sinus and localizing ligature or resection of the internal jugular vein are performed. When trauma penetrates the sinus during a mastoidectomy, the entire reaction of the body is different, and if the herd of infection in the mastoid is eliminated at the mastoidectomy, the forces comprised in what is termed resistance are protective factors, which minimize the danger of subsequently developing sepsis. Hence, I find myself fully in according with the views expressed by Klicpera. At the same time, it may not be amiss to remark that with average care sinal injuries are avoidable.

The annual meeting of the American Laryngological, Rhinological and Otological Society in 1939 concerned itself with the care of the patient after the surgical treatment for sepsis of otitic origin. Grove [57] stressed the need for maintaining the general comfort of the patient and indicated that the selection of the nurse was a most important factor in the after-treatment. The environmental room temperature preferably should be kept at 68 F., and a high caloric diet should be administered. Fluids should be forced, by mouth preferably but also by parenteral injections, to the end that the acidosis usually present in these patients is overcome. Sutherland [58] stressed the use of transfusion and emphasized that transfusion should not be considered a common routine procedure. Blood transfusions influence, either beneficially or adversely, the actual activities of the patient's blood, the bone marrow and the parenchymal organs. There should be a clear comprehension of the purpose sought by the transfusion, and the factors at issue need careful consideration before the transfusion is administered. For example, the transfusion may be needed to replace lost erythrocytes, in cases of anemia, or to restore to normalcy the blood plasma protein content and hemoglobin; or it may be used to dilute toxic blood. Galloway [59] contributes a paper on the use of chemotherapy in the postoperative stage of otitic sepsis. He emphasizes that chemotherapy should be

57. Grove, W. E.: Nursing, Feeding and Use of Parenteral Fluids, in Symposium on the Care of the Patient Following Operations for Sepsis of Otitic Origin, Tr. Am. Laryng., Rhin. & Otol. Soc. **55**:4, 1939.

58. Sutherland, J. M.: Use of Transfusions of Blood, in Symposium on the Care of Patients Following Operations for Sepsis of Otitic Origin, Tr. Am. Laryng., Rhin. & Otol. Soc. **55**:9, 1939.

59. Galloway, T. C.: Use of Chemotherapy, in Symposium on the Care of the Patient Following Operations for Sepsis of Otitic Origin, Tr. Am. Laryng., Rhin. & Otol. Soc. **55**:29, 1939.

employed in conjunction with other treatment and should not be used to replace therapy of known value and proved potency. Galloway warns that care should be exercised to ward off toxic reactions to drug therapy.

TECHNIC—A NEW PROCEDURE

Germán [60] reports an interesting case which he expertly handled in a new manner. Because the case report contains implications which evidently can be employed in cases of thrombosis of the cavernous sinus, I outline his case report separately. Reporting the case of a boy aged 13 years, who had mastoiditis, he states that several mastoidectomies, each revising a prior one, had to be performed. A week after the last of these, there developed retro-orbital pain, and the discharge from the mastoid wound increased. A week later, palsy of the abducens nerve, chills, fever and a profuse aural discharge were presented, to be followed by exophthalmos, ptosis, chemosis, somnolence and palsy. A diagnosis of petrositis and thrombosis of the cavernous sinus was then made.

The surgical treatment undertaken was original and interesting. The semicircular canals were skeletonized, and the dura of the middle and the posterior cranial fossae were exposed. The superior border of the petrosal pyramid was removed almost up to the internal auditory meatus, and the superior petrosal venous sinus was slit open for a distance of 2.5 cm. When the dura was lifted there escaped from the sinus a scanty amount of blood with a considerable number of red, purulent, degenerated pieces of thrombus. Free bleeding was not obtained. The symptoms of thrombosis of the cavernous sinus disappeared in the course of two weeks, and the patient recovered. On the basis of his satisfactory experience with the surgical procedure just described in this case, Germán suggests the use of this measure to obtain drainage in cases of thrombosis of the cavernous sinus.

LESIONS OF THE PARS PETROSA

The comprehension of the lesions, the establishment of a diagnosis of location and the surgical therapy for handling suppurations of the petrous pyramid have become almost stabilized. Therefore, it is to be expected that various refinements will ensue, and the record is commencing to denote this. Thus, Mangabeira-Albernaz [61] now turns his attention toward finding proper terms by which to designate the petrosal lesions. For example, he directs attention to the formation of terms. He states that when one forms a word, the beginning and the end of

60. Germán, T.: Ueber chirurgische Behandlung der Cavernosusthrombose (Ein Neues Operationsverfahren), Monatschr. f. Ohrenh. **73**:387, 1939.

61. Mangabeira-Albernaz, P.: Petrite e não petrosite, Rev. oto-laring. de São Paulo **6**:73, 1938.

it should have the same derivation. If the ending is a Greek element, such as "itis," the beginning should be of Greek derivation also. Therefore, "petritis" is a better term than "petrositis."

Rainer [62] made a histologic study of 200 temporal bones, cutting them in serial sections. He was interested in the development and the distribution of the cells in the petrous pyramid. He notes that the conditions of pneumatization in the apex differ from those in the mastoid process. The labyrinthine capsule, having attained its maximum growth at birth, thereafter no longer adds a factor to the growth of the base of the skull—which becomes enlarged by the addition of layers of bone to the upper surface of the pyramid. During the first year of life, this connective tissue bone is deposited in lamellar formation and is not rich in marrow spaces. Pneumatization of the mastoid process starts from the antrum, the hypotympanum and the eustachian tube. The progress of pneumatization follows the description given by Wittmaack, except in the region of the hiatus subarcuatus. Here connective tissue follows the path of the blood vessels in the petrous pyramid and fills the bone spaces up to the region of the periantral cells. In the periantral region the connective tissue meets the epithelial lining. The presence of the upper cell route and its extent depend on the width of periosteal bone between the superior semicircular canal and the dural plate. Pneumatization of the petrous pyramid starts between the ages of 4 and 6 years and may continue throughout life. Wilson,[63] in a study of the air cells of the petrous pyramid of a child of 13 years, reports that these air cells were well developed and that they arose from two different sources: first, from the extension of the mastoid cells and, secondly, from the tubal cells and the hypotympanum. The two systems of cells did not communicate with each other. The second group was closely related to the carotid canal and had a distinct channel, or opening, into the hypotympanum. Littenbauer [64] made an interesting study of the distribution of the cells. He took roentgenograms of the temporal bones of 50 cadavers, in both the Stenver and the Meyer position. After this, the bones were sectioned along their long axis. In all 50 pairs of bones, only 15 were found which presented well pneumatized petrous tips, and in only 1 instance were both bones alike. In 14 bones, peritubal cells were found. The author's most important observation was that whenever perilabyrinthine and apical cells were present, then the mastoid

62. Rainer, A.: Untersuchungen über die Entwicklung und den Ausbau der Pyramidenzellen, Arch. f. Ohren-, Nasen- u. Kehlkopfh. **145**:3, 1938.

63. Wilson, J. G.: The Air Cells in the Petrous Temporal Bone in a Child of Thirteen Years, Ann. Otol., Rhin. & Laryng. **48**:35, 1939.

64. Littenbauer, W.: Ueber die Pneumatisation des Felsenbeins. Eine anatomische und roentgenologische Studie über besonderer Berücksichtigung der konstitutionellen Verhältnisse, Ztschr. f. Hals-, Nasen- u. Ohrenh. **43**:314, 1938.

process was well pneumatized also. Gerlings[65] states that pneumatization of the perilabyrinth is more frequent than that of the apical region.

While these studies all have an abstract scientific value for the practicing otologist, they can be only of academic interest, since he is concerned with a problem which presents itself to him as a clinical entity. Littenbauer's study is important to the clinician, however, in evaluating symptoms in the development of petrous infections, because the surgeon will generally have at hand information as to the cellular content of the mastoid process and the extension of cellular structure into the perilabyrinthine region.

Tanaka,[66] from an examination of 14 temporal bones, reaches the conclusion that otitic meningitis is most often the result of infection of the cells of the pyramidal tip. He holds that this is true even when clinically the inflammatory process in this area shows evidence of spontaneous healing. Exactly how he reaches this conclusion is not clear to me, since it seems impossible to determine the degree of spontaneous healing that takes place in the cells of the tip, where meningitis subsequently develops. If the evidence from which he deduces this contention is obtained from a histologic examination of sections of the bone from the tip cell area, it must be recalled that similar bone tissue studied in specimens taken from patients with acute coalescent mastoiditis often reveals regions which are spontaneously healing, while the major part of the mastoid process is undergoing pathologic breakdown. Hence, this study can have only academic interest, and opinions as to the necessity of operative intervention for suppurative lesions in the cells of the tip cannot be based on Tanaka's findings.

Yoshié[67] reports a new method of demonstrating the petrosal apex roentgenologically. While the Stenver position shows the labyrinthine block of tissue and demonstrates the region of supralabyrinthine cells and the upper surface of the pyramid, information regarding the apex itself is not of much value, because of the distance between the anatomic structures of the apex and the film on which their shadows are recorded. Yoshié's technic gives a clearer picture of the apex, with a sharper contour of the carotid canal. The position is similar to that used for the Schüller or the Sonnenkalb technic. The patient's mouth is held widely open when the film is being made. The x-ray beam is directed slightly forward, to the external auditory canal of the ear nearest to

65. Gerlings, P. G.: Petrositis, Nederl. tijdschr. v. geneesk. **83**:504, 1939.

66. Tanaka, F.: Ueber die Bedeutung der Pyramidenspitzenentzündung für die Entstehung der otogenen Meningitis, Ausz. z. Otol. **42**:141, 1936; abstracted, Zentralbl. f. Hals-, Nasen- u. Ohrenh. **31**:76, 1938.

67. Yoshié, Y.: Röntgenologische Studien am Gehörorgan: III. Eine neue Methode zur röntgenologischen Darstellung der Pyramidenspitze, Ausz. z. Otol. **42**:47, 1936; abstracted, Zentralbl. f. Hals-, Nasen- u. Ohrenh. **31**:273, 1939.

the film. When the mouth is held widely open, the head of the condyloid process of the mandible becomes pushed forward beyond the posterior surface of the tuberculum articulare. Thus there is created a large space between the mastoid process and the ramus of the maxilla, facilitating the full projection of the structures of the petrous tip on the film or plate.

Among the case reports of particular interest, Gerlings [68] cites a case of petrositis in a girl 10 years old, in which the petrosal suppuration occurred in the third week of an attack of scarlet fever. One week subsequently palsy of the abducens nerve appeared, and then purulent meningitis developed. Gerlings utilized the Ramadier technic, and the child recovered.

This favorable outcome is gratifying, but it may be surmised that the significance of transient slight clinical manifestations was missed until paralysis of the abducens nerve and purulent meningitis became evident. Operation during the stage of petrosal involvement before meningeal infection has developed not only would give a greater assurance of recovery but would be a preventive measure, saving the patient the hazards to life which meningitis entails.

Towsen [69] reports a case of petrositis in a child of 9 years, in which the condition followed an antrotomy and removal of the bone covering the posterior and superior cranial fossae. There then developed palsy of the sixth nerve, pain along the distribution of the fifth nerve and meningitis. Fourteen days after the original operation a radical mastoidectomy was performed, and the cells of the petrous tip were removed because they were found to be diseased. This was done with a curet. The patient recovered. Rasmussen and Meyer [70] report on 8 cases of apicitis. In 7 the diagnosis was made clinically and by roentgen examination. In 1 the lesion was discovered only at autopsy. There were 2 deaths in this series. An important observation is made by these authors. In 5 of their cases, the lesion in the petrosa was combined with sphenoiditis. In this series, 3 patients made spontaneous recoveries, no operation having been performed on the apex. Jackson [71] performed an antrotomy on a 6½ year old girl. Three days after her discharge from the hospital, there were a rise in temperature, projectile vomiting and other clinical evidence of meningitis. Spinal tap yielded fluid that contained 6,000 cells; Streptococcus haemolyticus beta was present in the fluid. Admin-

68. Gerlings, P. G.: A Case of Petrositis with Purulent Meningitis Cured by Operation After Ramadier, Nederl. tijdschr. v. geneesk. **83**:2463, 1939.

69. Towsen, C. E.: Petrositis: Report of a Case with a Review of Some of the Literature, Arch. Otolaryng. **29**:468 (March) 1939.

70. Rasmussen, H., and Meyer, T.: Several Cases of Apicitis, Ugesk. f. læger **100**:1375, 1938.

71. Jackson, C. C. R.: Petrositis, Meningitis (Streptococcus Beta): Sulfanilamide, Drainage, Recovery; Report of a Case, Laryngoscope **48**:689, 1938.

istration of sulfanilamide and repeated spinal taps brought improvement in the picture of the meningitis within four days. Roentgenograms of the petrous pyramid, made in the Stenver position, revealed an apical abscess, which was drained by the subdural route. Cure resulted in two and a half months.

I am impressed by the long period of after-treatment and the interval of two and a half months required to effect a cure in this case. Perhaps, had the source of the suppuration in the petrosa been eradicated when the abscess was drained, the time required to procure healing would have been shorter. Danielewicz [72] performed translabyrinthine drainage in a case of apicitis with an accompanying subdural abscess. He found it difficult to maintain adequate drainage by the use of rubber drains and inserted a tracheotomy cannula, size 1, which proved adequate. In his case also healing required two and a half months. I can only direct attention to my comments on the prolonged time required to secure healing in the preceding case. In Danielewicz' case the same factors delayed healing. I may furthermore call attention to my comments in previous summations on the nonsurgical attempts to effect drainage in a direction against the force of gravity, upward from the petrosal bone into the subdural area. This case report speaks for itself and needs no further comment from me.

Mayer [73] reports 2 cases in which he operated, using the Ramadier technic. The cases are of special interest because of the fact that in both the pathologic condition occurred in the temporal bone which was less pneumatized. Mayer thinks that the reason for this is that in well pneumatized bone open spaces permit communication between the petrosa and the antrum and the middle ear, which suffices for drainage; but when faulty pneumatization exists such free communication spaces are less apt to be present or are entirely absent. In his second case, a parapharyngeal abscess developed. In another contribution Mayer [74] reported 4 more operations performed by the Ramadier technic. He substantiates an observation recorded by others, that the artery does not pulsate in the canal. I may add that the artery as it is seen in the canal has much the appearance of a vein. The outer coat is lost in the bony canal, and it is as easily compressible as is a vein of equal size. Mayer offers the suggestion that this route, namely that of the carotid artery into its bony canal, might be found useful as a channel for establishing drainage of the cavernous sinuses in cases of thrombosis.

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72. Danielewicz, J.: Use of a Tracheotomy Tube for Drainage in a Case of Apicitis with Subdural Abscess, Polski przegl. otol. **14**:587, 1938.

73. Mayer, O.: Zwei weitere durch Operation geheilte Fälle von Pyramidenspitzenabscess, Arch. f. Ohren-, Nasen- u. Kehlkopfh. **145**:349, 1938.

74. Mayer, O.: Fortschritte auf dem Gebiete der operativen Ohrenheilkunde (Freilegung der Arteria carotis interna), Wien. klin. Wchnschr. **51**:996, 1938.

Guillermin, Pesme and Coudane [75] report a case of latent petrositis in a young soldier who, without pain or discharge from the ear, had a swelling over the left mastoid process, accompanied by facial paralysis. Simple mastoidectomy revealed a coalescent mastoiditis, and after this surgical treatment the facial paralysis abated. Twelve days later, however, there were sudden attacks of nystagmus, and the patient died. The autopsy showed the dura to be adherent to the superior surface of the left pyramid, and there was a rupture of the bone over the internal auditory meatus, through which an apical abscess evacuated itself. In consideration of the extensive pathologic findings and the presence of a ruptured lesion in the apex, I cannot but suspect that here, too, the clinical signs and symptoms of the penultimate period between the mastoiditis and the ultimate meningitis were overlooked or not recognized for what they signified. The case is useful, therefore, with the others cited previously and 1 reported by Afonskij,[76] in stressing the importance of comprehending the symptomatology of the petrosal lesion per se.

Williams [77] reports a case of encapsulated empyema of the petromastoid. The patient had had bilateral otitis media, which lasted for a month, one year before the case came under Williams' observation. After the patient came under observation retroauricular pain and tenderness of the right side developed, and the patient complained of dizziness. Examination revealed a choking of the right disk, nystagmus to the left and paralysis of the right abducens nerve. A craniotomy was performed on the right side to save the eyesight, and there resulted a slight improvement in the clinical picture. Otoscopic examination showed a slight loss of hearing in the right ear. All caloric tests gave negative results. The result of roentgen examination was also negative. Mastoidectomy on the right revealed disease about the tegmen. Removal of the tegmen uncovered an epidural abscess. Working forward, the operator encountered and drained a large abscess in the apical region. Recovery ensued.

Iwaszkiewicz [78] reports 5 cases of inflammation of the cells in the petrosa. In 1 case this was accompanied by a cholesteatoma; in another, by purulent otitis media. In a case which terminated fatally, the formation of sequestrums involving the petrous tip took place and was accompanied by caries of the sphenoid. A retropharyngeal abscess developed

75. Guillermin, M.; Pesme, J., and Coudane, R.: Pétrosite latente, Ann. d'otolaryng., 1938, p. 729.

76. Afonskij, N. M.: A Case of Petrositis Involving the Pyramidal Apex, Vestnik otol. **1**:85, 1938.

77. Williams, H. L.: Encapsulated Empyema of the Petromastoid: Report of a Case, Ann. Otol., Rhin. & Laryng. **48**:236, 1939.

78. Iwaszkiewicz, J.: Ueber Entzündung der Pyramidenzellen, Polski przegl. otol. **13**:323, 1937; abstracted, Zentralbl. f. Hals-, Nasen- u. Ohrenh. **31**:378, 1939.

in 1 case, and in 1 case there was complicating sinus thrombosis. In all these cases operation was performed by the Eagleton technic.

Ionescu and his associates [79] report on a case of acute hematogenous osteomyelitis of the temporal bone in a girl 2 years of age, who suddenly was seized with chills and septic temperature, which lasted seven days. On the left side there appeared, from 7 to 8 cm. below the parietal suture, a fluctuating swelling. Rales developed in both lungs, and there were albuminuria, splenic enlargement and tympanites. Incision of the swelling on the skull yielded pus, which on culture showed Staphylococcus. Bare bone was encountered by the curet. Death followed twelve days later. At autopsy an osteomyelitic focus was found in the temporal bone, with a purulent thrombophlebitis of the lateral sinus and a meningoencephalitis. In all the organs of the body there were multiple abscesses. The author distinguishes three periods of this disease: (1) that of the local petrosal lesion, (2) that of the venous spread to the meninges and (3) that of general bacteremia.

In cases of osteomyelitis of the petrosquamomastoid, particularly in children, Laskiewicz [80] remarks, there is often found a swelling in the region of the malar bone, the eyelids and the neck. When the pyramid is also involved, the nerves from the fifth to the tenth inclusive become involved. Severe forms of this disease lead to meningitis, and it is spread through the canal of the internal carotid artery. In 2 of his cases sequestrums were found, with extradural abscesses and sinus thrombosis. He believes that early operation can stop the progress of the disease. It may be remarked that from even these few reports on osteomyelitic lesions of the petrosal pyramid, it is evident that the clinical picture they present differs sharply from what is presented in the average petrosal suppuration. Luckily this type of condition occurs much less frequently than the suppurations which develop in pneumatized bones. In the latter condition the picture is one of marked sepsis from the onset, and the thrombotic lesions in the veins and the metastatic foci in the rest of the body account for the sepsis. I have repeatedly drawn attention to the difference of the clinical pictures presented by these two differing lesions and also to the need of a different type of surgical treatment for each.

Lillie and Williams [81] report on chronic suppurative lesions of the petrous pyramid. They give new observations on these chronic lesions,

79. Ionescu, E.; Ionescu, A., and Dumitrescu, G.: Acute Hematogenous Osteomyelitis of the Temporal Bone, Rev. de ortop. şi chir. infant. **11**:17, 1938.

80. Laskiewicz, A.: Osteomyelitis of the Petrosquamomastoid in Children, Polski przegl. otol. **14**:599, 1938.

81. Lillie, H. I., and Williams, H. L.: Chronic Suppurative Lesions of the Petrous Pyramid: Report of Six Cases with Different Pathologic Characteristics, Arch. Otolaryng. **29**:345 (Feb.) 1939.

which have as yet not been fully recorded. Four of these cases are particularly noteworthy. In 1, there was a subacute otitis, which did not present an otorrhea. The appearance simulated that characteristic of mucosus otitis, with involvement of the fifth nerve and paralysis of the abducens nerve. At operation an abscess was located and subdurally drained. In another case, involving a man 34 years of age, who had a chronic purulent otitis media with complete loss of the membrana tympani, which had persisted since his childhood, paralysis of the seventh nerve appeared. On operation a caseous subdural abscess was located on the upper surface of the petrosa and was evacuated. A third case was that of a boy 12 years old, who had an acute purulent otitis media with mastoiditis, which spontaneously healed without surgical intervention. One and a half years later there appeared sudden ocular pain, headache and vomiting, followed by a profuse otorrhea. This patient died. At autopsy there were found a latent osteomyelitis at the base of the pyramid, thrombosis of the cavernous sinus, meningitis and a fistula into the lateral pharyngeal wall.

In the fourth case, a 30 year old patient with a chronic purulent otitis media had persistent headaches for a long time. A radical mastoidectomy was performed, and at operation a fistula was found behind the tubal orifice in the pharynx and another at the tubotympanic orifice in the middle ear.

Abstracts from Current Literature

Ear

IMPORTANT FACTORS IN THE CONSERVATION OF HEARING. H. B. HITZ, Wisconsin M. J. **38**:382 (May) 1939.

One type of progressive hearing defect, beginning usually in the late teens, is variously diagnosed as chronic dry catarrh of the middle ear, as hypertrophic and atrophic catarrh of the middle ear and by some, erroneously, as otosclerosis. Its characteristics are diminished air conduction and increased bone conduction of sound and observable bulging or retraction of the tympanic membrane. Various causes, such as hereditary diseases, diseases of early childhood, social diseases, malnutrition and endocrine disturbances, as well as deficiencies of blood and growth, all have advocates and critics.

Hitz believes that a large decisive element in the causation of this progressive diminution in hearing is the fixed and chronic habit that some persons have of blowing the nose violently, causing excessive alternate inflation and deflation of the middle ear. Usually some transitory obstructive lesion initiates the habit. Many writers have mentioned this factor of habitual pneumatic abuse. The cumulative effect of this incessantly repeated increasing of air pressure causes permanent changes in these delicate structures. Nature throws in more building material to strengthen against the physical strain, hence the impairment of hearing. Such effects of changing atmospheric pressure are had in an intensified form in airplane travel and cause a condition mentioned as "aero-otitis media acuta."

Treatment is chiefly preventive and consists in teaching children not to blow the nose forcefully. Both nostrils should be free when the nose is being cleared. Active treatment consists in breaking the habit and, on occasions, performing a minor surgical operation to increase ventilation.

The chronic snorer, continuously deflating his ears, is also often afflicted with regression of hearing. A correction of the nasal insufficiency improves this condition in some, and sleeping on the side rather than of the back leads to improvement in others.

JANNEY, Wauwatosa, Wis. [Am. J. Dis. Child.]

THE NUCLEI, CENTRAL PATHWAYS AND CONNECTIONS OF THE AUDITORY NERVE IN MAN. A. V. D. BROEK, Oto-rhino-laryng. internat. **23**:561 (Nov.) 1939.

The auditory nerve is composed of two main divisions, the cochlear and the vestibular, although the separation is not absolutely complete. After leaving the cochlear ganglion, the cochlear division has a communicating branch (vestibulocochlear branch of Oort) with the inferior vestibular ganglion and one with the saccular branch of the vestibular nerve (cochleosaccularis of Hardy). Weston stated the belief that this branch was not present in man. Scarpa's ganglion has two parts, the superior and inferior vestibular. To the former go the branches from the ampullae of the external and anterior canals and from the utricular macula; to the latter go branches from the macula of the saccule and the ampulla of the posterior canal. A communicating branch connects the saccule with the superior vestibular ganglion (Voit).

Fibers of the cochlear nerve terminate in the tuberculum acusticum and the ventral nucleus, some fibers going directly to the trapezoid body of the opposite side. From the two nuclei fibers decussate and enter the trapezoid body of the opposite side. Fibers from the trapezoid body ascend in the lateral lemniscus and, without interruption, terminate in the posterior corpora quadrigemina and the internal geniculate body. Other fibers go directly to the posterior quadrigeminate

body and the internal geniculate body of the same side. Some writers believe that fibers of the trapezoid body and of the lateral lemniscus are in connection with gray masses in the bulb: the superior olive, the nucleus of the trapezoid body and the nucleus of the lateral lemniscus. From the nuclei of the posterior quadrigeminate body and internal geniculate body a pathway goes to the superior temporal cortex, and a reflex pathway, by way of the posterior longitudinal bundle, may be followed to the anterior part of the spinal cord; it gives branches to the motor nuclei in the mesencephalon, metencephalon, myelencephalon and spinal cord.

The connections of the vestibular division are more complex. The fibers from the inferior vestibular ganglion terminate, according to Winkler, in the ventral nucleus (although most writers describe them as ending in the triangular nucleus), the nucleus of the descending bundle and Deiters' nucleus. It is probable that the saccular fibers decussate and give collateral fibers to the superior olivary nucleus and to the nuclei of the trapezoid body and the lateral lemniscus, thus connecting with the bulbar and spinal motor nuclei by way of the posterior longitudinal bundle. From the nucleus of the lateral lemniscus fibers go to the posterior quadrigeminate body and the internal geniculate body. Fibers from the superior vestibular ganglion go to Deiters' nucleus, although Winkler stated the belief that they go first to the triangular nucleus and the nucleus of the descending bundle. Deiters' nucleus is the principal origin of the vestibulospinal and the vestibulomesencephalic bundles. The vestibulospinal bundle descends through the bulb to the gray matter of the anterior horn of the spinal cord, where the medial group of motor cells sends fibers to the dorsal muscles of the trunk and a lateral group sends fibers to the ventral muscles and to the extremities. The vestibulomesencephalic bundle ascends and sends fibers to the third, fourth, fifth, sixth and seventh nuclei. A third group of fibers from Deiters' nucleus and from the triangular nucleus and the nucleus of the descending bundle form the juxta restiform body and terminate in the fastigii and globosus nuclei and the cortex of the flocculus. Cerebellifugal fibers go from the cortex of the flocculus to Deiters' nucleus; axons of cells of the roof nuclei form the fastigiovestibular tract and terminate in Deiters' nucleus of both sides. According to Winkler, some fibers from the roof nuclei and the triangular nucleus join the cerebellorubral bundle, go by way of the superior peduncle, decussate through the commissure of Wernekink and terminate in the red nucleus of the opposite side. Cerebellipetal pathways are:

1. The dorsal cerebellospinal bundle of Flechsig, the ventral cerebellospinal bundle of Gowers and the direct cerebellospinal bundles. Flechsig's bundle arises in the column of Clark in the dorsal and lumbar regions and goes, by way of the restiform body, to the cortex of the superior vermis. Gowers' bundle originates in the central gray substance of the cord, decussates, rises through the bulb, twines around the superior cerebellar peduncle and terminates in the cortex of the vermis. The direct cerebellospinal bundles conduct excitations of deep sensibility to Purkinje's cells.

2. From cells of the principal and accessory olivary nuclei, axons of the olivocerebellar bundle decussate, go through the inferior cerebellar peduncle and terminate in the cerebellar cortex of the opposite side.

3. Fibers from the nuclei of Goll and Burdach join Flechsig's bundle.

4. Vestibulocerebellar fibers have already been described.

5. There is a crossed connection between the cerebral cortex and the cerebellar cortex through the pontile nuclei. The frontopontile, parietopontile and temporopontile bundles arise in the cerebral cortex, pass through the internal capsule and mesencephalon and terminate in the pontile nuclei. Axons from the cells of the pontile nuclei cross the midline, follow the middle peduncle and end in the cortex of the vermis and of the cerebellum. They form the moss fibers around the small granular cells; axons from the latter cells terminate around Purkinje's cells.

Cerebellifugal pathways have already been described in part. Axons of Purkinje's cells enter the white substance and terminate around the central nuclei, especially the dentate nucleus. The cerebellorubral-thalamic bundle arises in the dentate nucleus and the nucleus globosus and terminates in the red nucleus and the external nucleus of the thalamus on the opposite side. Axons of the cells of the red nucleus become the rubrospinal bundle, which crosses at Forel's decussation and may be followed as far as the sacral cord. Fibers of the rubrospinal, posterior longitudinal, predorsal and vestibulospinal bundles enter the gray substance of the spinal cord in the anterior horn and terminate around the groups of motor cells for the muscles of the extremities.

DENNIS, San Diego.

POSITIVE FISTULA SYMPTOM DUE TO CHEMICALS. ERICH RUTTIN, Pract. oto-rhino-laryng. **2**:293 (Sept.) 1939.

The author reports 2 cases of chronic suppurative otitis media (history of radical mastoidectmy in 1) associated with granulations, which were treated with a 5 to 10 per cent solution of silver nitrate. There immediately appeared vertigo and nystagmus on the same side. These symptoms were not exhibited if the usual fistula test was made or if at the region of the granulations dry cotton applicators applied hydrogen peroxide, alcohol, tincture of iodine, glycerin or paraffin. The author believes that the phenomenon elicited is purely chemical, presupposing minute erosions of the labyrinthine wall, too small for response to the air pressure test yet large enough to permit a penetration of the silver nitrate solution; by diffusion through the fluid, the silver nitrate causes a turgescence of the neuroepithelium of the copula, which produces the vestibular reaction.

PERSKY, Philadelphia.

OTITIC THROMBOPHLEBITIS OF THE CAVERNOUS SINUS. WALTER MAHN, Ztschr. f. Hals-, Nasen- u. Ohrenh. **45**:109, 1939.

The author reports 3 cases of thrombosis of the cavernous sinus following otitic infections. The route of extension in the first case was from the superior petrosal sinus. In the second case it followed also thrombosis of the superior petrosal sinus, which was apparently associated with suppuration in the petrous apex. The third case followed thrombosis of the inferior petrosal sinus, which was a sequel to thrombosis of the jugular bulb.

The author's purpose in presenting these cases is to emphasize the fact that on occasion the cavernous sinus may be directly involved from otitic suppurations by way either of the superior or of the inferior petrosal sinus.

PERSKY, Philadelphia.

INDICATIONS FOR LABYRINTHECTOMY. OTTO MAYER, Ztschr. f. Hals-, Nasen- u. Ohrenh. **45**:117, 1939.

Mayer discusses the difficulties that face the otologist in deciding on the indications for labyrinthectomy. To clarify these indications he has grouped the usual symptoms as follows: (1) labyrinthine stimulation (nystagmus, vertigo, vomiting and loss of equilibrium); (2) labyrinthine destruction (deafness and no responses from the labyrinth); (3) circumscribed destruction of the bony labyrinthine wall (fistula), and (4) meningitis.

While the symptoms of group 1 indicate a labyrinthitis, their presence does not necessarily mean that the labyrinthine operation is in order. Group 2 suggests a "dead" labyrinth. Group 3 indicates a viable labyrinth, as evidenced by a positive reaction to the fistula test. Group 4 speaks for itself in that the meningitis is usually of otitic origin.

Dependence on any one of these groups of symptoms as a positive indication for a labyrinthectomy is fallacious. Consequently, the author has outlined the following indications as a positive index showing when to operate: (1) the presence

of symptoms of all four groups; (2) the involvement of three groups (1+2+3, 1+2+4, 2+3+4, 1+3+4); (3) the involvement of any two groups (1+2, 1+3, 1+4, 2+3, 2+4 and 3+4). By grouping the symptoms in this fashion, he simplifies the problem and provides a splendid guide to the otologist in deciding on the indications for labyrinthectomy.

PERSKY, Philadelphia.

DEAFNESS DUE TO OSTEOMYELITIS. GERHARD THEISSING, Ztschr. f. Hals-, Nasen- u. Ohrenh. **45**:284, 1939.

The author reports a case of deafness in a 43 year old patient, which appeared in the course of osteomyelitis of the long bone. The deafness appeared first in one ear and then, after a remission, in the opposite ear. Later both ears were involved, total deafness finally resulting. Throughout the course there was at no time evidence of suppuration.

He believes that the pathologic condition was a toxic degeneration of the eighth nerve and that in his particular case labyrinthitis was not a factor.

PERSKY, Philadelphia.

RECOVERY AFTER THROMBOSIS OF THE LATERAL SINUS, BULB AND INTERNAL JUGULAR VEIN. OPERATION BY GRUNERT'S TECHNIC. TENSHI RIN, Oto-rhino-laryng. **12**:817 (Oct.) 1939.

Rin records a case of recovery in a 30 year old man in whom thrombosis of the lateral sinus, jugular bulb and internal jugular vein developed after an acute exacerbation of chronic mastoiditis on the right side. On September 25 the patient caught cold and was generally indisposed. On September 28 he had marked pain in the right mastoid area, which in a week became quiescent. From October 17 to 24 he had numerous chills and fever. On November 4 he was admitted to the hospital. His right membrana tympani showed a small perforation in the lower posterior quadrant, and only a slight amount of otorrhea was noted. He retained the hearing in the right ear, 45 per cent for low tones and 70 to 90 per cent for high tones. The left ear was normal. There was no pain or tenderness over the right mastoid. There was nothing remarkable about his neck except one or two palpable cervical nodes on each side, and the examiner was led to believe that the infection might be other than otologic in origin. Roentgen examination, however, was suggestive of bony destruction of the plate of the sinus. Queckenstedt's sign was positive on the right. On November 9 exploration of the right mastoid process was made. The superficial cortex was normal. On removal of the plate of the sinus, the venous wall was found coated with granulation tissue, and pus welled out from the cells next to the jugular bulb. When the vascular channel was opened, the lateral sinus was found to be completely thrombosed, and pus was found within the jugular bulb. The operation was completed by removing the venous wall and by ligating the internal jugular vein.

On November 14, after the first operation, the septic temperature persisted and required a revision of the wound. The operation disclosed thrombosis extending down to the level of the lower border of the cricoid cartilage. The vessel was completely resected, and the bony wall of the jugular bulb was also removed after Grunert's technic. Two days after the operation the patient's temperature came down, and he was dismissed from the hospital forty-two days after admission.

Rin emphasizes the importance of early recognition of the presence of thrombosis, and when the extensive thrombus occurs in the lateral sinus, involving the bulb and internal jugular vein, the safest procedure is the removal of the entire thrombosed vascular wall together with the structure about the jugular bulb.

HARA, Los Angeles.

HEARING TESTS AND HEARING AIDS: A CLINICAL AND EXPERIMENTAL STUDY. LENNART HOLMGREN, Acta oto-laryng., 1939, supp. 34.

This monograph, issued as a supplement to the *Acta oto-laryngologica* and published in English, is much too long for a critical abstract. It is, however,

reported in this form because it is felt that it is worth the careful study of every otologist who is interested in hearing aids and the problems of the hard of hearing. The monograph thoroughly covers the whole field of functional testing, including a statement of the fundamental acoustic principles, an evaluation of speech tests, an evaluation of conversation and whisper tests and their disadvantage, an assay of practical value of tuning fork tests from an acoustic standpoint, a critical survey of present day audiometer construction, a comparison of the value of the tuning fork tests and audiometric examinations, a critical survey of the practical value of the customary hearing aids, a critical survey of the various ways of prescribing and trying out hearing aids, a description of a new audiometer of the author's own and an exhaustive bibliography. It is regretted that space does not permit an exhaustive abstract of this excellent and important contribution.

GROVE, Milwaukee.

Pharynx

NEW CONCEPTS OF THE FUNCTIONS OF THE TONGUE. MAX A. GOLDSTEIN, Laryngoscope **50**:164 (Feb.) 1940.

After reviewing the classic teaching about the vital function of the tongue in speech production, that the tongue is absolutely essential in the production of various consonant elements, and describing the mechanics of the production of the elements of articulate speech, Goldstein reports 3 cases of amputation of the tongue, in each of which the patient was able after convalescence to produce oral speech that was understandable and distinct. He showed that the tongue is not an indispensable organ, that various other parts of the oral cavity may be utilized to substitute for the tongue in the complicated mechanism of understandable fluent speech and that knowledge of the physiology of the tongue as an organ of speech is subject to revision.

The article includes descriptions of various special functions of the tongue in animals.

A phonographic recording of the speech of a tongueless man is described.

WOOD, Newark.

RETROMOLAR ACCESSORY SALIVARY GLANDS. H. LEPP, Monatschr. f. Ohrenh. **73**:695 (Nov.) 1939.

A retromolar accessory salivary gland is described, and a number of instances are cited in which such a finding is discussed. This accessory gland may give rise to a tumor growth, as it was found by the author to have done in a 66 year old woman who had a tumor behind the inferior third molar tooth on the left side. This tumor was resected, and the histologic observations are evaluated so as to prove that the origin of the tumor was connected with this accessory gland. One photograph and eleven photomicrographs accompany the article.

LEDERER, Chicago.

Larynx

LARYNGECTOMY: A PLEA FOR A NARROW FIELD OPERATION. CHEVALIER JACKSON, Surg., Gynec. & Obst. **70**:517 (Feb.) 1940.

The author strongly advocates a modification of the standard technic for laryngectomy in what he terms the "narrow field" laryngectomy. Having observed that the "wide field" laryngectomy presents many disadvantages, such as shock, trauma, impaired blood supply, difficulties with flaps, air pocketing, large wounds, increased flow of serum, retarded healing and greater risk of pharyngostoma, he feels that there are certain definite indications for a revised technic. These are present principally in cases in which the malignant disease of the larynx is still

intrinsic and of limited extent, yet too far advanced to warrant laryngofissure. The author is cognizant of the fact that a narrow field laryngectomy is inadvisable: (1) when the lesion is extrinsic either by origin or extension; (2) with an involvement of the internal perichondrium, even though it may be only inflammatory; (3) when there is an impairment of the arytenoid mobility; (4) with any evidence of lymphatic involvement, even if only inflammatory; (5) when there is a high degree of malignancy of the growth as indicated histologically, and finally (6) when there are the usual contraindications for any operative procedure. He advocates that the larynx be removed through a midline incision without the use of flaps and that no search for metastatic involvement of the lymphatics be made when there are no nodes palpable, for here the lymph channels can be sealed by subsequent irradiation. However, when the lymph nodes are palpable, he believes that the case is one for irradiation rather than for laryngectomy. He stresses the extreme importance of early diagnosis of cancer of the larynx.

He then outlines his technic of operation and the postoperative care and, in conclusion, again suggests that in the early stages the narrow field laryngectomy should be the method of choice.

PERSKY, Philadelphia.

CATARRHAL ESOPHAGITIS. S. BALINOFF, Monatschr. f. Ohrenh. **2**:76 (Feb.) 1940.

The diagnosis of catarrhal esophagitis is usually overlooked, as the symptoms are generally attributed to other organs. Modern methods now permit one to diagnose this disease in living subjects. The most common causes are influenza, gastric disturbances and infectious diseases. The postmortem examination of patients with disturbances of the esophagus revealed pathologic conditions, although the patients had never had any previous internal complaints. Esophagoscopy usually reveals increased secretion, hyperemia and swelling of the mucous membrane. The patient complains of a burning sensation when food passes. This is usually localized behind the sternum. The tongue is coated and the temperature slightly increased, and roentgen examination shows diminished motility, rarely spasm. A semisolid diet is advised and belladonna administered. The author wishes to suggest the possibility that esophagospasm might be preceded by catarrhal esophagitis and is therefore caused by it.

LEDERER, Chicago.

ABACILLARY LARYNGEAL TUBERCULOSIS. FRIEDRICH LÜTGERATH, Ztschr. f. Hals-, Nasen- u. Ohrenh. **45**:316, 1940.

The author discusses the occurrence of tuberculous lesions in the larynx in the absence of evidence of tubercle bacilli in the sputum. He admits that, while an associated tuberculous lesion in the lungs facilitates a correct diagnosis, difficulties arise when tuberculous changes in the chest are either minimal or absent.

He made a study of 52 patients, who, though the sputum was not found to contain organisms, were considered to have tuberculosis of the larynx. The analysis included comparisons of blood counts with emphasis on leukocyte count, differential studies and determination of the sedimentation rates. The sites of involvement in their order of frequency are the ventricular bands, the vocal cords, the posterior wall of the larynx, the epiglottis, the arytenoids and, finally, the aryepiglottic folds. A further analysis showed that the lesions were found to be extrinsic in 4 cases, both extrinsic and intrinsic in 13 cases and solely intrinsic in 35 cases. He also attempted to classify the lesions from the standpoint of a possible root of infection and found that there was a hematogenous dissemination from the lungs in 10 cases; in 32 cases, the lungs showed a productive nodular cirrhotic type of tuberculosis, while in 10 cases, the lungs were normal. Attempting to establish a relationship between the site of the lesion and the type of pulmonary disease, he found that the intrinsic laryngeal involvement is most frequently associated with the nodular cirrhotic pulmonary lesions, while the hematogenous group showed a greater tendency to a more general involvement, particularly extrinsic.

In summarizing the results of treatment, he reported 25 complete cures and improvement in 23 cases; 4 patients became progressively worse. Four months after the institution of treatment, 41 patients were discharged from the hospital as either cured or markedly improved. PERSKY, Philadelphia.

Nose

A NEW VIEW OF THE PNEUMATIZATION OF THE SINUSES. K. BERNFELD, Pract. oto-rhino-laryng. **2**:276 (Sept.); 337 (Nov.) 1939.

Assuming the Wittmaack theory of pneumatization of the mastoid process as a basis for his deductions and studies, the author attempted to establish a similar relationship between inflammatory processes in the nasal mucous membrane of both infants and younger children and the normal rate and extent of pneumatization of the sinuses. In an analysis of a group of children between the ages of 8 and 14 years, who were troubled with marked adenoid hypertrophy, roentgen examination did not reveal any appreciable influence on the size and shape of the sinuses, as compared with those of children who had not had any of these hyperplastic recurrent infections. He analyzed another group of patients who suffered from acute or chronic rhinitis (from birth or early infancy, with or without adenoid hypertrophies), syphilis, diphtheria or ozena, and in these patients too could find no evidence of an influence on the rate of pneumatization of these sinuses.

The author notes Eagleton's dictum that pneumatization in postfetal life depends primarily on the rate of change of the red bone marrow to the white marrow. Hence, all those processes, whether of an inflammatory or of another nature, which have a tendency to protract or restrain the red bone marrow state and so prevent the formation of the white bone marrow, cause a disturbance or retardation in the rate of pneumatization.

The author's impression is that certain factors can influence the variability in both size and extent of the sinuses: (1) hereditary influences, (2) the rate of change of the red to yellow bone marrow, which can be affected by both morphologic and physiologic factors, (3) the correlated influences of all adjacent bones (not merely the characteristics of the individual bone containing the sinuses) and (4) certain exogenous factors.

Furthermore, the author stresses the hormonal factor, describing, and placing special emphasis on, the role of the pituitary gland. Since the secretion of this gland exerts a definite influence on all growth and bone formation, it is perhaps one of the more important endogenous factors that can affect both the type (or form) of pneumatization of the sinuses and the activity of the hypophysis. He describes the extensive pneumatization found in certain forms of acromegaly, cerebrohypophysial obesity, hypergenitalism and other disturbances of a similar nature. The influence of the thyroid and of the pineal gland is also stressed, but to a lesser degree. An interrelationship of all these three glands, however, is necessary to regulate growth. Hence, these intrinsic factors, either individually or in association with some extrinsic factor, play a direct role in the rate and extent of pneumatization of the sinuses. Furthermore, the variability in the formation of the sinuses is affected through two influences of hormonal activity, (1) on regulation of growth and (2) on the mucous membrane epithelium.

In summarizing, he states: 1. The variability of the pneumatization of the sinuses is a direct consequence of a manifold process associated with the development of the bones of the skull. 2. Hereditary factors play a decisive role in regard to constancy as an individual variation. 3. Inflammatory conditions of the nasal mucosa do not exert any appreciable influence on the degree of pneumatization of the sinuses. 4. The normal process of development of the sinuses and the rhythm of pneumatization are strongly influenced by dysfunction of the endocrine glands. 5. Hence it is probable that physiologic variations in pneumatization are directly influenced by endocrine functions. PERSKY, Philadelphia.

Miscellaneous

TRANSPORT OF AIR ALONG SHEATHS OF PULMONIC BLOOD VESSELS FROM ALVEOLI TO MEDIASTINUM: CLINICAL IMPLICATIONS. CHARLES C. MACKLIN, Arch. Int. Med. **64**:913 (Nov.) 1939.

From an experimental study on animals and from clinical observations of the postmortem findings in human beings, the author was able to demonstrate that air leaks from the pulmonary alveoli usually spread along the perivascular sheaths. When this pulmonic, interstitial emphysema occurs, it readily extends up to the roots of the lungs and there produces symptoms by a pneumatic pressure on the larger blood vessels. A small amount of air in this region can produce considerable pressure. Thus, the symptoms are often out of proportion to the amount of air leakage. Should this air leakage be continued, it may rupture and produce a pneumomediastinum.

The author discusses the symptoms and the clinical applications, stressing the fact that the disease may arise as a sequel to an atelectasis or to an increased intrapneumonic pressure (as during a faulty administration of an anesthetic by the closed method). The extension of air along the pulmonary vessel sheaths causes primarily an embarrassment of the pulmonary circulation. Should infection follow the break and leakage, suppuration may occur along this route, and, finally, there may be a formation of subpleural blebs, which in turn may rupture and produce a pneumothorax. With a pneumomediastinum, there is another train of symptoms, as emphasized by generalized subcutaneous emphysema or even pneumopericarditis.

The prognosis, particularly of a pneumomediastinum in an uninfected patient, is usually not so grave as has been previously assumed. When the amount of air is relatively low, it can be absorbed in two weeks. However, when there is a continuation of the leak and there seems to be no opportunity for complete resorption, other methods of treatment should be instituted. The author advocates an incision into the neck and an insertion of tubes into the mediastinum, plus the application of suction. For controlling the leak he recommends an artificial pneumothorax, which compresses the lung and acts as a buffer. PERSKY, Philadelphia.

TREATMENT OF PNEUMOCOCCIC MENINGITIS WITH SULFAPYRIDINE AND ITS SODIUM SALT. HORACE L. HODES, HARRY S. GIMBEL and GEORGE W. BURNETT, J. A. M. A. **113**:1614 (Oct. 28) 1939.

Hodes, Gimbel and Burnett report their experience with the use of sulfapyridine and its sodium salt in a series of cases of pneumococcic meningitis. Previous to using this drug they had a mortality rate of almost 100 per cent. All the 29 patients treated between 1930 and 1936, when no specific therapy was used, died. From December 1936 to October 1938 17 patients were treated with sulfanilamide, but only 1 recovered. Of a total of 46 patients treated between January 1930 and October 1938, but 1 recovered.

From October 1938 until May 1939 they treated 17 patients with pneumococcic meningitis, using, in addition to all the tested procedures, sulfapyridine and its sodium salt. They have been gratified by a striking reduction of the mortality with the use of this drug, 8 of the 17 patients (47 per cent) having completely recovered.

Their technic is as follows: The oral administration of sulfapyridine is instituted on admission. In addition, intravenous therapy with a 5 per cent solution of sodium sulfapyridine in distilled water is begun. An initial dose of 0.1 Gm. per kilogram of body weight is given intravenously every six hours until the patient shows definite clinical signs of improvement and until two successive lumbar punctures produce sterile fluid. After this, the sodium sulfapyridine is given only twice a day for several days longer and then its use is discontinued. Intravenous therapy should be resumed at the first indication that a relapse has occurred.

Sulfapyridine given by mouth is absorbed irregularly and only in limited amounts. Intravenous injection of its sodium salt at regular intervals hastens its concentration in the blood and spinal fluid. The concentration of free sulfapyridine in the spinal fluid should be maintained at a level of from 10 to 15 mg. per hundred cubic centimeters.

Though some toxic symptoms have been encountered, such as granulocytopenia, nausea, vomiting, cyanosis and hematuria, none of these was fatal in any of the cases.

GORDON, Philadelphia.

OBSERVATIONS ON THE PATHOLOGY OF MÉNIÈRE'S SYNDROME. C. S. HALLPIKE and H. CAIRNS, Proc. Roy. Soc. Med. **31**:1317 (Sept.) 1938.

A description is given of the histologic changes in the temporal bones of 2 subjects with Ménière's syndrome. In each case, the patient died shortly after surgical section of the vestibular nerve, and in each the following pathologic changes were found in the affected temporal bone: (1) gross dilatation of the endolymphatic system affecting chiefly the scala media of the cochlea and the saccule and (2) degenerative changes in Corti's organ and the stria vascularis.

It is considered unlikely that these changes in the tissue represent the results of a serous labyrinthitis due to toxins which reached the labyrinth through the round window membrane or via the blood stream from some focus of infection. The view is expressed that the endolymphatic dilatation is in fact brought about by a failure of the absorption mechanism of the endolymph and is supported by the disclosure of certain fibrotic changes in the perisaccular connective tissue described by Guild.

A possible mechanism is suggested correlating the histologic changes with the clinical features of the disease. According to this the attacks of labyrinthine disorder are due to anoxemic paralysis of its end organs induced by sudden rises of intralabyrinthine pressure.

In general, the observations are considered to support the clinical opinions of Crowe and of Wright, that the morbid condition should be regarded as a specific disease of the labyrinth.

HALLPIKE and CAIRNS.

OBSERVATIONS ON A CASE OF "MÉNIÈRE'S" DISEASE. C. S. HALLPIKE and A. J. WRIGHT, Proc. Roy. Soc. Med. **32**:1646, 1939.

The histologic changes are described in the temporal bones of a patient with "Ménière's" disease who died six months after the first attack of vertigo.

As in the 2 cases previously described by Hallpike and Cairns, the affected temporal bone showed fibrotic changes in the perisaccular connective tissue, with dilatation of the scala media.

The observations are considered to support the view previously put forward, that this combination of changes represents the essential morbid anatomy of a specific disease of the labyrinth.

HALLPIKE and WRIGHT.

CAISSON DISEASE. A. C. REGHIZZI, Arch. ital. di otol. **51**:408 (Aug.) 1939.

The two current theories of the causation of caisson disease are the theory of gas embolism and the hydraulic pressure theory. Reghizzi discusses especially the effects of the disease on the auditory and respiratory apparatus and reports 3 illustrative cases. Of the numerous studies of the pathologic changes of caisson disease, those of Panse and of Vail are noted. Panse found the labyrinthine vessels filled with bloody grumous material and surrounded by perivascular infiltration, the cochlear fibers detached from the bone by hemorrhagic exudate and, in places, hemorrhages in the scala tympani. Vail's experimental work on dogs revealed in all cases copious hemorrhages in the submucosa of the middle ear and in the vestibule. In Magnotti's experiments on rabbits, he found exfoliation of

the epithelium of the nose and larynx, which in the latter formed plugs that occluded more or less the tracheal and the bronchial lumen. The prognoses of the accidents of decompression vary according to the part of the ear involved. Lesions of the middle ear range from simple tympanic congestion to purulent otitis media. For the most part, these lesions disappear within a few weeks without leaving a significant trace. The labyrinthine lesions in some cases regress, even though slowly; in others they persist and are characteristically associated with a state of disequilibrium and deafness. Labyrinthine examination is especially important, since otoscopic examination often shows no appreciable lesion. In some cases the hearing returns more or less completely, while in others the unilateral deafness, tinnitus and vertigo are not ameliorated. From a medicolegal point of view, the incapacity for work is increased and the patients not only will be unable to resume their former occupations but will be unable to accomplish work requiring notable effort.

DENNIS, San Diego, Calif.

NEW SURGICAL TREATMENT OF VARICOSE VEINS OF THE OESOPHAGUS. CLARENCE CRAFOORD and PAUL FRENCKNER, Acta oto-laryng. **27**:422, 1939.

The authors call attention to the submucous varices so often seen in the cardiac area of the esophagus in cases of splenomegaly, especially when splenectomy has been done. They conceived the idea of collapsing these bleeding veins and varices by injecting quinine hydrochloride and ethyl carbamate (urethane). A special long injection needle was devised by Frenckner, which was used through a 9 mm. Jackson esophagoscope. In four successive treatments the varices were collapsed by the injection of the solution of quinine hydrochloride and ethyl carbamate, the operators beginning with the uppermost varices and working down the esophagus in the successive treatments. No hemorrhage was occasioned by the injections, and a control esophagoscopy one month after the last treatment failed to disclose any remaining varices.

GROVE, Milwaukee.

THE USE OF VENTRICULOGRAPHY FOR OTOGENIC AND RHINOGENIC ABSCESSES OF THE BRAIN. Y. MEURMAN, Acta oto-laryng. **27**:509, 1939.

Meurman calls attention to the use of ventriculography for suspected brain abscess. This method of examination may be particularly helpful in cases in which the dura does not indicate a fistula or stalk. Changes in the ventriculogram can be expected only when the abscess has developed to sufficient size to influence the shape or volume of the ventricle. Meurman's technic is to make two trephine openings, one on each side, about 2.5 cm. from the sagittal suture and just above the coronary suture. The dura is incised to avoid damaging vessels on the surface of the brain with the cannula. Both cannulas are inserted at the same time. About 30 to 40 cc. of fluid are removed from the two sides and an equal volume of air insufflated. The cannulas are left in situ while films are made. Immediately after the films are exposed the insufflated air is allowed to escape.

The changes in the ventriculogram with otogenic and rhinogenic abscesses consist of dislocation, incomplete filling with air, in some cases a total lack of air in a part of the lateral ventricle or in the whole ventricle and sometimes a deformation in the shape of the ventricle. He emphasizes the fact that abscesses in the temporal lobe and even those in the occipital lobe can dislocate the anterior horns to the healthy side. Meurman deprecates the use of ventriculography for suspected cerebellar abscess as being dangerous and not likely to give any particular information.

GROVE, Milwaukee.

Book Reviews

Les maladies du larynx: Clinique et thérapeutique. By G. Canuyt, professeur de clinique oto-rhino-laryngologique à la Faculté de Médecine de Strasbourg, with the assistance of M. Truffert, ancien chef de clinique à Faculté de Médecine de Paris, oto-rhino-laryngologiste de l'Hôpital Bon-Secours de Paris and M. Tarneaud, oto-rhino-laryngologiste de l'Hôpital Bellan et du Conservatoire National de Musique de Paris, and with the cooperation of Charles Wild, ancien chef de clinique à la Faculté de Médecine de Strasbourg, oto-rhino-laryngologiste de policlinique à l'Hôpital Civil de Strasbourg. Price, 270 francs. Paris: Masson et Cie, 1939.

The foreword presents the associate contributors, among whom are Professor Ségura, of Buenos Aires, who wrote a chapter on grippal laryngitis, and Dr. Robert Rendu, of Lyon, who wrote an interesting chapter on laryngocele, its diagnosis and treatment. The introduction is unusual, as it discusses laryngology of the past, the present and the future. Treating the historical background, discussing the accomplishment of present methods in diagnosis and treatment and speculating about the future of laryngology, it prepares the reader for a pleasant study of the remainder of the book.

The volume is in three parts. The first is devoted to the examination of the larynx. The chapter on mirror laryngoscopy is well presented, and the one on direct laryngoscopy is by Haslinger himself. Only his own method is considered. The distally lighted instrumentarium of Jackson is ignored. The only instrument shown is one of the first esophagoscopes, devised thirty-five years ago by Jackson. Curiously, no comparison is made with the modern distally lighted laryngoscope of Jackson and the present proximally lighted laryngoscopes of Haslinger. The chapter on indirect laryngoscopy is the only one that is not interesting and in keeping with present medical usage. Roentgenography of the larynx is well presented, and special emphasis is laid on lateral roentgenograms of the neck. Tomography is also discussed, and excellent roentgenograms made by the tomographic method are shown.

The second part takes up pathology of the larynx. Syphilis and tuberculosis of the larynx are handled well, with full bibliographic references. Polyps and papillomas are presented as benign tumors. A valuable discussion on cancer of the larynx is given and presents the author's views on partial and total laryngectomy. It is remarkable that the author states that in 75 per cent of the cases of cancer of the larynx in his series the growth originated in the laryngopharynx. In the United States statistics prove that in about 75 per cent of cases of cancer of the larynx the lesion originates as intrinsic cancer within the larynx. The usual methods and results in the use of radium and high voltage roentgen therapy are shown. The section on tracheotomy is well illustrated, and the methods of treatment and the results in cases of laryngeal stenosis are set forth. For the removal of foreign bodies in the larynx the Haslinger technic and instrumentarium are advocated; when this method has failed, the author has had recourse to external operative removal by cricothyroidotomy and tracheotomy. One feels that the use of a modern distally lighted instrument might have prevented the necessity of external operative removal of the laryngeal foreign bodies in the cases cited. Acute laryngitis in children is discussed at length; particularly interesting are the observations of Le Mée, André Bloch and Maurice Bouchet. Chronic laryngitis and *laryngite sèche* (atrophic laryngitis) are each given a chapter. Infiltrative laryngitis, perichondritis and traumas of the larynx are considered. A study of lesions of the laryngeal nerves completes the second division.

The third part is devoted to diseases of the voice and includes discussion of examination of the larynx and the voice in cases of disturbances of endocrine

glands, difficulties with the change of voice (*mue*), psychic aphonia and disphonia, difficulty with the singing and speaking voice, nodules of the vocal cords, the voice in persons with recurrent paralysis and the voice after the removal of the larynx. The chapter on the last-named subject is particularly interesting to physicians and laryngologists who are working in phonetics and adds a great deal to the volume.

The book is acceptable in every way and should be in the library of every laryngologist.

News and Comment

CASSELBERRY PRIZE FUND

The Casselberry Prize Fund of the American Laryngological Association has available $150, to be given as a prize award for original work in rhinology and laryngology. Theses or reports of work done must be submitted before Feb. 1, 1941. Further particulars may be obtained from Dr. Charles J. Imperatori, secretary of the American Laryngological Association, 108 East Thirty-Eighth Street, New York.

Directory of Otolaryngologic Societies *

NATIONAL

American Medical Association, Scientific Assembly, Section on Laryngology, Otology and Rhinology

Chairman: Dr. LeRoy A. Schall, 243 Charles St., Boston.
Secretary: Dr. Louis H. Clerf, 1530 Locust St., Philadelphia.
Place: Cleveland. Time: June 2-6, 1941.

American Academy of Ophthalmology and Otolaryngology

President: Dr. Frank Brawley, 30 N. Michigan Ave., Chicago.
President-Elect: Dr. Frank R. Spencer, Physicians' Bldg., Boulder, Colo.
Executive Secretary: Dr. William P. Wherry, 1500 Medical Arts Bldg., Omaha.

American Broncho-Esophagological Association

President: Dr. Gabriel Tucker, 250 S. 18th St., Philadelphia.
Secretary: Dr. Paul Holinger, 1150 N. State St., Chicago.

American Laryngological Association

President: Dr. Gordon Berry, 36 Pleasant St., Worcester, Mass.
Secretary: Dr. Charles J. Imperatori, 108 E. 38th St., New York.
Place: Atlantic City, N. J. Time: May 28-30, 1941.

American Laryngological, Rhinological and Otological Society, Inc.

President: Dr. J. Mackenzie Brown, 1136 W. 6th St., Los Angeles.
President-Elect: Dr. James A. Babbitt, 1912 Spruce St., Philadelphia.
Secretary: Dr. C. Stewart Nash, 708 Medical Arts Bldg., Rochester, N. Y.
Place: Ambassador Hotel, Los Angeles. Time: June 16-18, 1941.

SECTIONS:

Eastern.—Chairman: Dr. N. S. Weinberger, Robert Packer Hosp., Sayre, Pa.
Place: Philadelphia. Time: Jan. 10, 1941.

Southern.—Chairman: Dr. William G. Kennon, Doctors Bldg., Nashville, Tenn.
Place: Nashville, Tenn. Time: Jan. 8, 1941.

Middle.—Chairman: Dr. Walter H. Theobald, 307 N. Michigan Ave., Chicago.
Place: Chicago. Time: Jan. 27, 1941.

Western.—Chairman: Dr. Robert C. Martin, 384 Post St., San Francisco.
Place: San Francisco. Time: Feb. 1-2, 1941.

American Otological Society

President: Dr. George M. Coates, 1721 Pine St., Philadelphia.
Secretary: Dr. Isidore Friesner, 36 E. 73d St., New York.
Place: Atlantic City, N. J. Time: May 26-27, 1941.

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* Secretaries of societies are requested to furnish the information necessary to keep this list up to date.